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THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health.

The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, papers of scientific and clinical interest which are presented at the annual meetings of that society, as well as articles submitted direct to the Editor. Obviously opinions expressed do not necessarily represent those of the Editor or the American Academy of Periodontology.

THE AMERICAN ACADEMY OF PERIODONTOLOGY

1959 Annual Meeting—September 10, 11, 12, 1959

New York City, New York

SPECIAL ISSUE NEXT

We believe the next issue of the Journal of Periodontology will be of special interest to all of our readers. Plans are now almost complete, and it is expected the issue will be in the mails in late July or early August. The last issue this year will not be published until after the Academy Meeting, so it will probably be mailed in late October, or early November.

A. W. WARD, D.D.S.

San Francisco, California

Dr. A. W. Ward of San Francisco, California has been active in the field of periodontology for over a half a century. During this time he has earned much renown for his many clinics, papers and movies. He is also well-known for his development of new instruments and materials used in periodontology. No one in this country is a more enthusiastic promoter of the field of periodontics than Dr. Ward.

Dr. Blanquie in writing about Dr. Ward said:

"His development of instruments permitting a simple and definite approach to the surgical treatment of periodontal lesions revived and brought into popular use therapeutic principles that were known for centuries; through his application of a cemental dressing to protect the exposed alveolar areas surrounding the teeth the surgical periodontal treatment became humane and practical. The ingredients of this dressing form the basis of all similar dressings in use at the present time."

THE JOURNAL OF PERIODONTOLOGY

APRIL, NINETEEN HUNDRED FIFTY NINE

Periodontal Reaction to Functional Occlusal Stress

by SIGURD P. RAMFJORD, L.D.S., M.S., PH.D.*

CHARLES A. KOHLER, D.D.S., M.S.*, *Ann Arbor, Michigan*

THE reaction of the periodontium to occlusal trauma has been studied in various laboratory animals[1, 6, 18, 21, 23]; furthermore, a few experimental investigations have been reported in humans[8, 12, 18]. The main source of histologic information regarding the adaptation of the periodontium to a variation in occlusal stress has been human autopsy material[2, 3, 4, 7, 10, 11, 12, 13, 14, 15, 17]; however, the clinical history of occlusal stress of most of the autopsy material has been very inadequate. Therefore there is still much that can be learned about the effects of occlusal stress upon the human periodontal tissues by studying material in which the clinical history of occlusal stress is known and the functional occlusal forces have been at least partially controlled.

The purpose of this paper is to present some observations of periodontal adaptation to a known functional increase and decrease in occlusal stress. The study is limited to the labio-cervical area and coronal one half of the root of fifteen anterior teeth in 14 humans.

MATERIAL

The material utilized in this study was obtained also for a study of the healing of the periodontium following gingival flap operations. A description of the procedures that were used to obtain the material has been submitted for publication[9]. Block sections, including gingival and other periodontal structures, and about one half of the root of the experimental tooth, were removed surgically from individuals who were scheduled to have complete dentures in at least one of the dental arches. About 4 to 12 millimeters of the labial alveolar process along the root was included in each specimen (Fig. 1). Detailed clinical examinations of the teeth and periodontal conditions were repeated at the various stages of the experiment. Pictures and roentgenograms were taken of all the patients.

CLINICAL FINDINGS

The clinical information has been compiled in Table I. Initially some of the patients had several posterior teeth missing, and an attempt was made at that

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TABLE I

Case No.	Age of Patient	Experimental Tooth	Occl. Stress Prior to Experiment	Gingival Condition	Days of Incr. Occl. Stress	Days Without Antagonists	Days After Flap Oper.
1	18	mand. r. cuspid	normal	mild gingivitis		14	14
2	16	max. r. central	heavy	mild gingivitis		14	28
3	34	max. l. cuspid	very heavy (bruxism)	severe gingivitis		15	28
4	27	max. r. cuspid	heavy	mild gingivitis	85		85
5	58	max. l. cuspid	very heavy	gingival atrophy	88		88
6	48	max. r. cuspid	normal	normal gingiva		98	94
7	27	max. r. cuspid	heavy	periodontitis	106		105
8	68	max. r. cuspid	very heavy (mobility 1)	mild gingivitis		106	112
9	68	max. l. lateral	very heavy (mobility 1)	mild gingivitis		106	112
10	55	max. r. lateral	normal	slight ging. atrophy		75	122
11	34	max. r. cuspid	heavy	gingivitis		146	138
12	37	max. r. lateral	normal	mild gingivitis	145		145
13	57	max. r. cuspid	heavy	mild gingivitis	148		145
14	64	max. r. lateral	no antagonist	mild gingivitis		169	162
15	50	max. r. lateral	very heavy	normal gingiva	198		196

time to evaluate and classify the functional occlusal stress on the experimental teeth. Occlusal relations, posterior occlusal support, mobility during functional movements, attrition, and radiographic findings served as a basis for classification of the occlusal stress as: 1) normal, 2) heavy, 3) very heavy.

Following the initial examination all the posterior maxillary teeth were extracted in 6 of the 14 patients in preparation for immediate dentures. It was assumed that this complete loss of posterior support would increase the functional load on the remaining anterior teeth. In 9 of the experimental teeth all of the teeth of the opposing jaw were extracted in preparation for dentures, thereby leaving the experimental tooth without an antagonist. The patients did not wear any opposing dentures during the period of observation, and the only functional stimuli the experimental tooth could receive would come from biting against the opposing alveolar ridge and from lip and tongue function.

The number of days between the flap operation and the removal of the specimens are recorded in Table I because bone

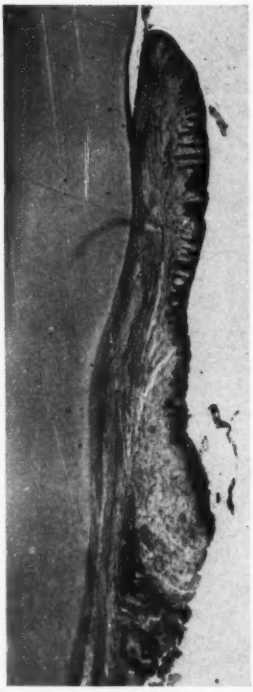

Fig. 1. Photomicrograph of a section from a typical specimen. Case 10. Maxillary right lateral incisor. Magnification X8.

changes on the labial aspect of the alveolar process and the alveolar crest should be related to the trauma of the muco-periosteal flap procedure.

HISTOLOGICAL FINDINGS

Case Number 1

Rapid formation of new bone is evident at the alveolar crest and along the periodontal membrane side of the alveolar bone. Large cuboidal osteoblasts practically cover the entire inner surface of the alveolar bone. Only a few rather heavy coarse Sharpey's fibers enter the bone with 1-5 osteoblasts between the individual fiber bundles. The alveolar process is fairly thick, and the labial surface is smooth without any indication of bone apposition or resorption. On the surface of the root a narrow zone of

Fig. 2A. Note the distinct layer of new bone on the periodontal membrane side of the alveolar bone. Case 2. Magnification X75.

precementum can be observed. The cementoblasts are small and the cementoblastic activity seems to be very slow. The alveolar crest fibers and the principal periodontal fibers exhibit a well defined functional arrangement, but under high magnification early atrophy and degeneration of the principal periodontal fibers is apparent in small focal areas in the middle of the periodontal membrane. The collagen fibers appear partially disintegrated and replaced by reticulum-like fibers in an edematous intercellular substance.

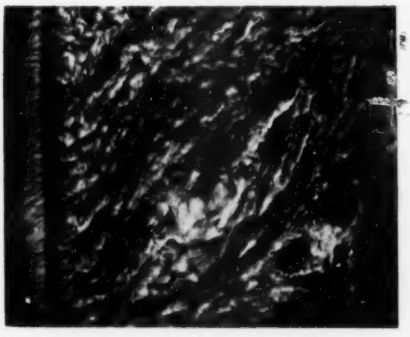

Fig. 2B. Azan stains for collagen. Numerous Sharpey's fibers entering the cementum on the left side of the picture. Heavy bundles of fibers entering the alveolar bone on the opposite side. Case 2. Magnification X800.

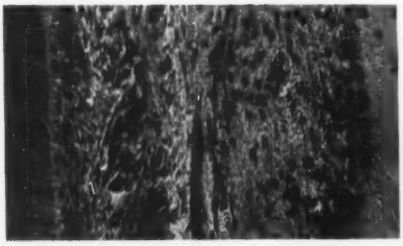

Fig. 2C. Photomicrograph of H & E stained section from same specimen as Figs. 2A and B. Well defined layer of precementum. Beginning degeneration and loss of functional pattern of collagen fibers. Magnification X800.

Case Number 2

Like in case number 1 there is evidence of rapid osteogenesis at the alveolar crest and along the periodontal membrane surface of the alveolar bone (Fig. 2 A). Relatively few heavy bundles of Sharpey's fibers are seen entering the alveolar bone. The labial surface of the alveolar process appears rough. The alternating areas of resorption and repair are possibly related to surgical trauma at the time of the flap operation 28 days prior to the removal of the specimen. The resorptive activity may also be related to the rebuilding that takes place in the alveolar process following loss of antagonists and the apposition of alveolar bone. A great number of small Shar-

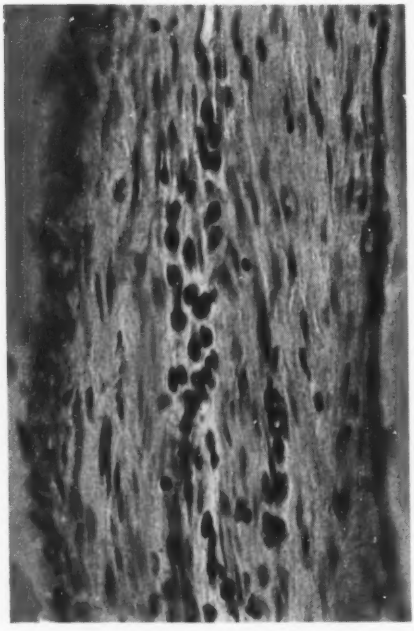

Fig. 2D. Plasma cells and lymphocytes in the middle of the periodontal membrane. Collagen fibers parallel with the surfaces of the tooth and the alveolar bone. Case 2. Magnification X650.

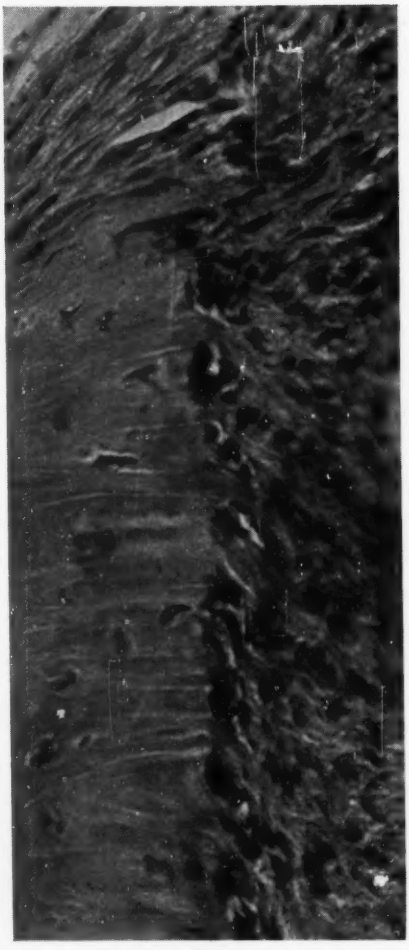

Fig. 3A. High magnification of the alveolar crest and the attachment of Sharpey's fibers. Case 3. Magnification X800.

Fig. 3B. Resorption of bone on the labial side of the alveolar process and newly formed bone on the periodontal membrane side of the alveolar bone. Dilated blood vessels. Periodontal fibers mainly parallel to the surface of the bone and the root. Actual width of the periodontal space: 0.08 — 0.10 mm. Case 3. Magnification X275.

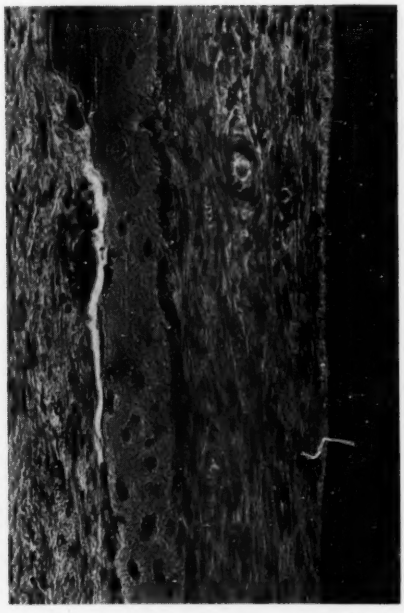

Fig. 3C. From same specimen as Figs. 3A and B. Close to the area where the alveolar bone plate is missing. Primitive type of new bone formed in an attempt to bridge the described defect in the labial aspect of the alveolar process. The Collagen fibers are parallel to the surface of the bone and the root. Magnification X275.

pey's fibers can be seen entering the cementum (Fig. 2 B), and a well defined layer of precementum is present on the root surface (Fig. 2 C). The alveolar crest fibers are arranged in a well defined functional pattern, and most of the principal fibers are made up of normally appearing collagenous fibers. Only in small areas in the middle of the periodontal membrane can beginning degeneration of the collagen fibers be observed (Fig. 2 C). A few inflammatory cells (Fig. 2 D), mainly plasma cells, appear in a scattered distribution extending from the bottom of the gingival crevice into the periodontal membrane proper for a distance of 3-4 millimeters apically to the alveolar crest.

Case Number 3

Rapidly progressing osteogenesis can be observed at the alveolar crest and along the periodontal membrane surface of the alveolar bone (Fig. 3 A). Bone resorption is evident on the labial aspect of the alveolar process (Fig. 3 B). The labial bone plate of the alveolar process is absent from a point 5 millimeters apically to the free gingival margin to the border of the specimen for a distance of 3 millimeters. On the root surface corresponding to the area of missing alveolar bone, resorption has extended into the dentin and the defects have been only partially repaired by deposition of new cementum. A narrow zone of precementum is present on the remainder of the root surface, but only in focal areas can cementoblastic activity be observed with certainty. The alveolar crest fibers exhibit a well defined functional arrangement. The attachment of the principal fibers appears to be much weaker to the alveolar bone than to the cementum. Small areas of slight degenerative changes

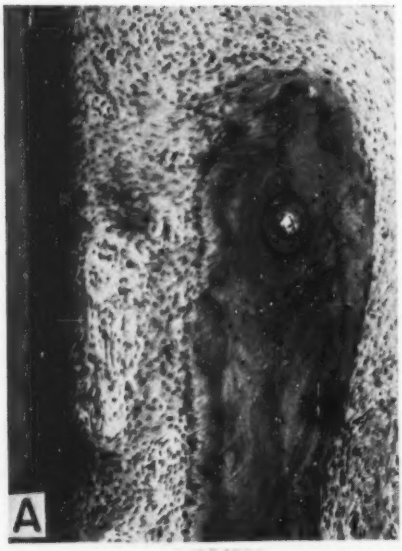

Fig. 4A. Newly formed bone at the margin of the alveolar crest and along the surface of the alveolar bone. Actual width of periodontal space in crestal area: 0.15 — 0.20 mm. Case 4. Magnification X266.

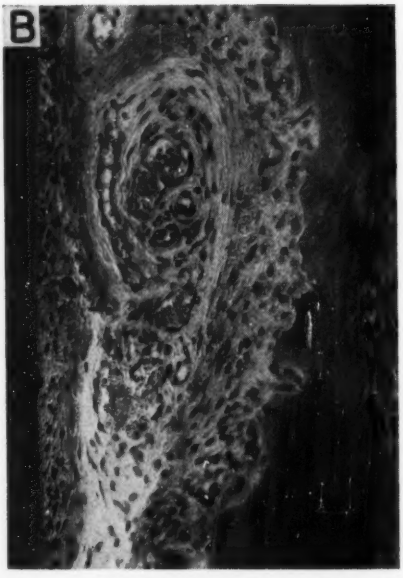

Fig. 4B. Resorption of the alveolar bone. Vascular granulation tissue in the periodontal membrane. Note intact fibers parallel to and attached to the surface of the cementum. Case 4. Magnification X310.

can be found in the periodontal membrane. The principal periodontal fibers are arranged mainly parallel to the surface of the root and the alveolar bone (Fig. 3 C). A few plasma cells and lymphocytes are present in the periodontal membrane, especially in the areas of degeneration.

Case Number 4

Osteogenesis is evident at the margin of the alveolar crest (Fig. 4 A). Several areas of active resorption and repair can be observed on the periodontal membrane side of the alveolar bone (Fig. 4 B). On the labial aspect of the alveolar process there is evidence of fairly rapid osteogenesis, but the osteoblasts derived from the periosteum of the alveolar process are much smaller and more squamous in character than the osteoblasts on the periodontal membrane side of the alveolar bone. Numerous cementoblasts and a well defined zone of precementum can be observed on the root surface. There is a marked widening of the periodontal space in the cervical area. The functional pattern of the collagen fibers is well defined, especially in the area of the alveolar crest. The oblique fibers run nearly parallel with the surface of the tooth and alveolar bone. There are numerous large vascular channels present in the periodontal membrane, and areas of vascular granulation tissue can be seen (Fig. 4 B).

Case Number 5

Apposition of new bone is evident on the labial aspect of the alveolar crest and the alveolar process (Fig. 5 A). On the periodontal membrane side the alveolar bone is undergoing resorption. The labial plate of the alveolar process is very thin, and in some areas there has been loss of the continuity of the alveolar process because of resorption from the periodontal membrane side of the alveolar bone. Many areas of resorption can be observed on the root surface. In some places the resorption extends into the dentin. Very little repair can be

Fig. 5A. The entire alveolar crest is made up of newly formed bone. Root resorption extending into the dentin. Case 5. Magnification X97.

Fig. 5B. High magnification (X280) from Fig. 5A. Compression of the periodontal membrane (actual width 0.06 mm.). The fibers are arranged parallel to the surface of the tooth and the alveolar bone. Active resorption of the alveolar bone.

found in the areas of root resorption. There is a marked compression of the periodontal membrane (width 0.06 mm.) corresponding to the root resorption (Fig. 5 B). This patient appears to have a marked tendency for resorption of cementum and dentin corresponding to compression and increase in pressure within the periodontal membrane. A large number of dilated blood vessels engorged with blood can be observed in both the free and attached gingiva (Fig. 5 C). The reason for this increase in blood supply is not apparent from our sections. The clinical picture is included to show the gingival recession and the heavy occlusion (Fig. 5 D).

Case Number 6

Several layers of newly formed bone are found on the periodontal membrane side of the alveolar bone (Fig. 6). Resorption of bone can be observed on the labial side of the alveolar process. There is fibrosis of the bone marrow. A well defined layer of precementum is evident on the root surface. The alveolar crest fibers exhibit a well defined functional pattern. In the remainder of the periodontal membrane the fibers are oriented parallel to the root surface. Focal areas of mucoid degeneration can be seen in the central part of the periodontal membrane. The mild chronic gingival inflammation extends into the periodontal membrane, slightly apical to the alveolar crest, where a focal area of inflammation apparently has produced a notch in the alveolar bone.

Case Number 7

The alveolar crest appears very thick and a slow apposition of new bone is evident at the crestal margin. On the periodontal membrane surface of the alveolar

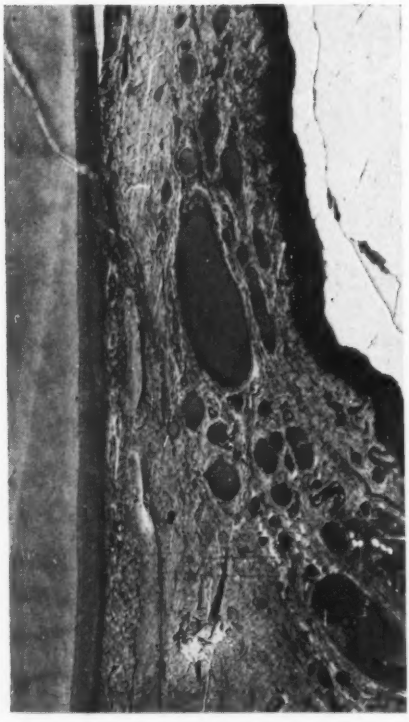

Fig. 5C. Large dilated blood vessels with a thin endothelial lining seen in the free and attached gingiva. No apparent connection with the compressed periodontal membrane. Case 5. Magnification X31.

Fig. 5D. Clinical picture of case 5 immediately prior to the removal of the specimen. Gingival recession and heavy occlusion with spreading of the maxillary anterior teeth.

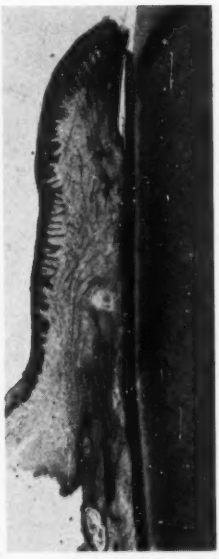

Fig. 6. Notch in the alveolar bone, slightly below the alveolar crest, associated with granulation tissue and inflammatory cells. Only mild gingival inflammation. Case 6. Magnification X10.

bone alternating areas of resorption and repair can be observed. The bone marrow is slightly fibrotic. A distinct layer of precementum is present on the root surface. The alveolar crest fibers are heavy and they exhibit a well defined functional pattern. The horizontal and oblique periodontal fibers have a very compact functional arrangement which closely resembles the tendon-like character of the periodontium in dogs. Only a few blood vessels are present in the periodontal membrane, and a very limited amount of interstitial loose connective tissue can be observed.

Case Number 8

Apposition of new bone is progressing at a rapid rate on the alveolar crest and along the periodontal membrane side of the alveolar bone. There is evidence of active bone resorption on the labial surface of the alveolar process. The bone marrow is partially fibrotic. Only a narrow zone of precementum can be observed. The cementoblasts are few and squamous in char-

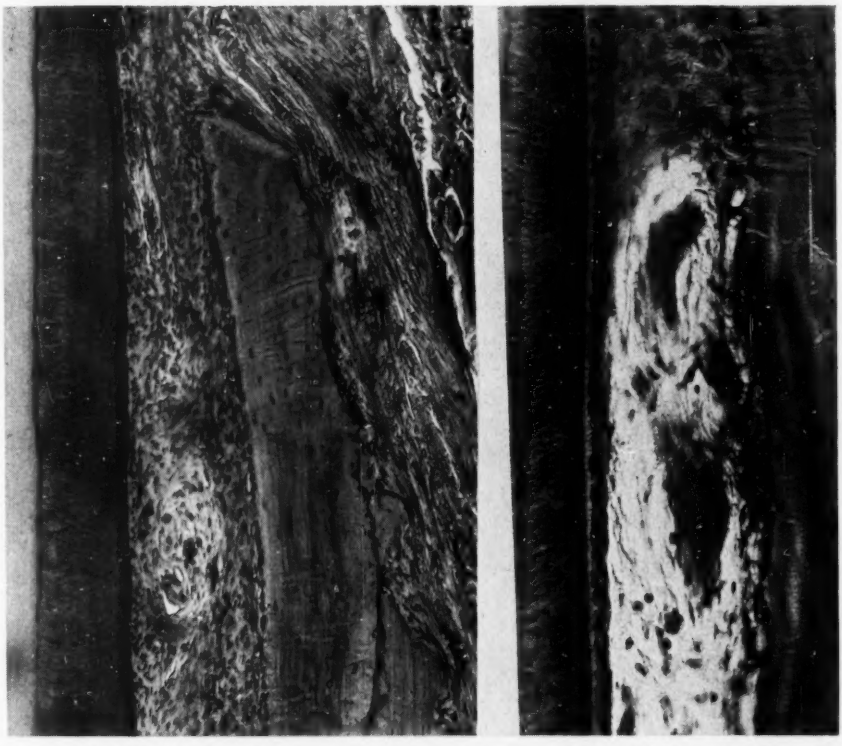

Fig. 7. (Left). Newly formed alveolar crest. Instead of normal horizontal fibers, the fibers are oriented parallel to the surface of the root and the alveolar bone. Oblique fibers can be seen at the bottom of the photomicrograph. Case 9. Magnification X170.

Fig. 8. (Right). Mucoid degeneration and a few lymphocytes and plasma cells in the periodontal membrane. Note the well defined layer of fibrous connective tissue on the surface of the root and the undisturbed pattern of the Sharpey's fibers, while the area of degeneration extends to the surface of the alveolar bone. Actual width of the periodontal space in this area: 0.12 mm. Case 11. Magnification X210.

acter in contrast to the numerous large osteoblasts and the wide zone of newly formed bone on the surface of the alveolar bone. A functional arrangement of the alveolar crest fibers is apparent, but there is evidence of beginning degeneration of the principal periodontal fibers with focal areas of mucoid degeneration and a few lymphocytes and plasma cells. In other areas within the periodontal membrane young granulation tissue is replacing the collagenous periodontal fibers. This granulation tissue is characterized by numerous newly formed vascular channels. The old collagen fibers have very long and narrow spindle shaped nuclei, and even the nuclei exhibit a slightly wavy pattern. Under high magnification one can observe a large number of fine Sharpey's fibers attached into the cementum, and there is absolutely no evidence of degeneration or atrophy along the root surface. The distance between the individual bundles of Sharpey's fibers is much greater on the alveolar bone surface than on the cemental surface. But the individual fiber bundles entering the bone is much thicker than the fibers that enter the cementum.

Case Number 9

Apposition of new bone is evident at the alveolar crest and along the periodontal

A
B
C
D

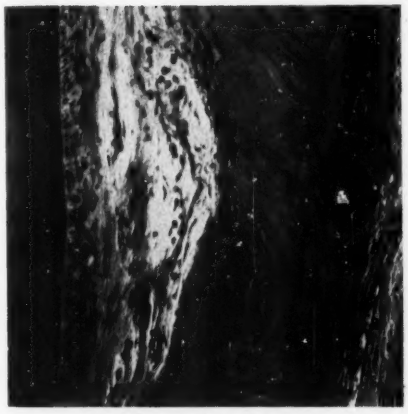

Fig. 9E. High magnification (X250) of area 3 mm. below the alveolar crest from same case as Figs. 9A, B, C, D. Mild mucoid degeneration of the periodontal membrane associated with slight resorption of the alveolar bone. Note the intact root surface and Sharpey's fibers. A few lymphocytes and plasma cells in the area of degeneration. Apposition of bone on the labial surface of the alveolar process.

membrane side of the alveolar bone (Fig. 7). Resorption is seen on the labial surface of the alveolar process. A fairly wide zone of precementum is present on the root surface, but only a few active cementoblasts can be seen. Most of the collagen fibers of the periodontal membrane run parallel to the root surface. There is evidence of diffuse areas of beginning mucoid degeneration of the periodontal membrane. In one such area of mucoid degeneration, about 5 millimeters apically to the alveolar crest, a considerable number of inflammatory cells are found.

Case Number 10

A wide zone of newly formed bone can be observed at the alveolar crest. There also is evidence of new bone formation along the periodontal membrane side of the alveolar bone. Active resorption is found on the labial side of the alveolar process. The labial bone plate which constitutes a fusion between the alveolar bone and the cortical bone on the alveolar process is very thin and without marrow spaces. A well defined zone of precementum is present on the root surface, and by careful study one can observe numerous areas of resorption and complete repair of the cementum. The alveolar crest fibers exhibit a well defined functional pattern. The remainder of the periodontal membrane is rather vascular, and there is evidence of beginning disuse atrophy and degeneration. Old collagenous fiber bundles characterized by very narrow long spindle shaped cells still have a functional orientation, while the young fibroblasts appear parallel to the root surface or frequently without any pattern of functional arrangement.

Case Number 11

Active bone apposition can be observed at the alveolar crest and along the periodontal membrane side of the alveolar bone. There is evidence of bone resorption on the labial side of the alveolar process. A relatively wide zone of precementum is apparent on the root surface. The periodontal fibers exhibit a functional arrangement at the alveolar crest. Areas of mucoid degeneration (Fig. 8) and a few lymphocytes and plasma cells can be seen scattered throughout the periodontal membrane. Most connective tissue cells of the periodontal membrane are arranged parallel to the root surface.

Fig. 9A. Alternating areas of resorption and apposition of alveolar bone. Slight hyalinization of the periodontal fibers. Intact fibers and attachment on the surface of the cementum. Actual width of periodontal space: 0.09 — 0.10 mm. Case 12. Magnification X157.

Fig. 9B. The alveolar bone plate is absent in the lower one-third of the photomicrograph. Case 12. Magnification X13.

Fig. 9C. Area of resorption extending into the dentin. Higher magnification (X78) of the area of missing alveolar process Fig. 9B. Muscle attachment into periosteum-like periodontal membrane.

Fig. 9D. Slight hyalin degeneration in the middle of the periodontal membrane and resorption at the alveolar crest. Different section from same area as Fig. 9A. Magnification X250.

Case Number 12

Lamellated newly formed bone is present on the labial aspect of the alveolar crest and along the labial surface of the alveolar process. Alternating areas of resorption and repair can be seen on the surface of the alveolar bone (Fig. 9 A). The labial bone plate is missing for a distance of 3 millimeters (Fig. 9 B) starting 4 millimeters apically to the alveolar crest and extending 7 millimeters apically to the alveolar crest where the alveolar bone again is present. Corresponding to this area of missing alveolar process, there is evidence of root resorption which in some instances extends into the dentin (Fig. 9 C). A well demarcated zone of precementum can be observed on the root surface. There is compression of the periodontal membrane in the cervical area with evidence of slight hyalin degeneration in the middle part of the periodontal membrane (Fig. 9 D). Mucoid degeneration can be found in other areas of the periodontal membrane (Fig. 9 E). The periodontal membrane has the appearance of periosteum where the labial bone is missing. The connective tissue cells are arranged parallel to the surface of the root and the muscle fibers are attached to this periosteal-like periodontal membrane.

Fig. 10A. Compression of the periodontal membrane (actual width 0.08 — 0.09 mm.). Slight hyalinization in the periodontal membrane. Alternating resorption and apposition of alveolar bone. Case 13. Magnification X182.

Fig. 10B. Photomicrograph from about 4 mm. below the alveolar crest. The same specimen as Fig. 10A. Fusion between labial cortical bone and alveolar bone. Actual width of the periodontal space in this area: 0.13 — 0.15 mm. Magnification X100.

Case Number 13

At the labial aspect of the alveolar crest there is evidence of new bone formation (Fig. 10 A). Alternating areas of resorption and apposition of bone can be observed on the periodontal membrane side of the alveolar bone. The alveolar process is thick and alternating areas of resorption and new bone formation can be observed on its labial surface. The marrow spaces are very small, and the line of fusion can be observed between the cortical layer of the alveolar process and the alveolar bone (Fig. 10 B). There is moderate fibrosis of the bone marrow. A very narrow zone of precementum can be seen under high magnification. There is no evidence of cemental resorption. The periodontal membrane appears slightly compressed at the alveolar crest, and beginning hyalinization can be observed in the middle of the periodontal membrane. Close to the area of hyalinization, the blood vessels appear enlarged. The collagen fibers in the rest of the periodontal membrane appear very compact and they exhibit an easily recognizable pattern.

Fig. 11A. Rapid apposition of bone at the alveolar crest and along the inner surface of the alveolar bone. Resorption on the labial surface of the alveolar process. Width of periodontal space: 0.10 — 0.14 mm. Case 14. Magnification X150.

Fig. 11B. High magnification (X347) of the periodontal membrane 3-4 mm. below the alveolar crest from the same case as Fig. 11A. Beginning disorganization of the periodontal fibers in the central portion of the periodontal membrane. Note osteogenesis and cementogenesis.

Case Number 14

Marked osteoblastic activity can be observed at the alveolar crest and along the periodontal membrane side of the alveolar bone (Fig. 11 A). Resorption is taking place on the labial side of the alveolar process. A well defined layer of precementum is present on the root surface. Beginning atrophy and degeneration of the periodontal fibers can be recognized in part of the periodontal membrane (Fig. 11 B), but well defined bundles of oblique periodontal fibers are still present. A few plasma cells and lymphocytes are found in focal areas of mucoid degeneration. Numerous evenly distributed small Sharpey's fibers enter the cementum, while only a few heavy bundles of Sharpey's fibers are inserted into the alveolar bone.

Case Number 15

Resorption is evident at the margin of the alveolar crest. Alternating areas of resorption and repair can be observed on the periodontal membrane surface of the alveolar bone (Fig. 12 A), while new bone formation is taking place on the labial aspect of the alveolar process. The labial bone plate is very heavy and made up of almost solid bone with only a few small marrow spaces present. A well demarcated zone of precementum can be observed on the root surface, and there is no evidence of cemental resorption. There is evidence of compression of the periodontal membrane at the alveolar crest (width 0.1 mm.) and beginning hyalinization of periodontal fibers in this area. Beginning hyalinization of the periodontal membrane is evident also several millimeters apically to the alveolar crest. Relatively large blood vessels with a thick endothelial lining can be seen at the alveolar crest and at the periphery of other areas of hyalinization. The periodontal fibers at the alveolar crest appear folded and compressed. In the periodontal membrane the fibers are arranged parallel to the root surface. The clinical picture of this case (Fig. 12 B) is included to illustrate the occlusal relations.

DISCUSSION OF FINDINGS

The histological findings were similar in the eight maxillary specimens where the opposing teeth were extracted. In all of these cases a remodeling of the alveolar process could be observed associated with a lingual incisal movement of the teeth. Apposition of bone appeared at the alveolar crest and along the periodontal membrane surface of the alveolar bone, while resorption was evident on the labial aspect of the alveolar process. The layer of alveolar bone that apparently had been deposited after the extraction of the opposing teeth was measured microscopically and the measurements are listed in Table II.

TABLE II

Case No.	*Days Without Antagonist*	*Apposition of Alveolar Bone in Millimeters*
2	14	0.05—0.07
3	15	0.06—0.08
10	75	0.10—0.12
6	98	0.11
8	112	0.15—0.20
9	112	0.14—0.17
11	146	0.08—0.10
14	169	more than 0.25

In case number 14 the entire labial bone wall in our specimen consisted of new bone, so the thickness of the newly deposited bone could not be measured, but it must have exceeded the present thickness of the alveolar process (0.25 mm.). Other cases showed complete replacement of the crestal portion of the alveolar process with new bone following the loss of the antagonist. Of course, it cannot be stated with absolute certainty that these measurements were obtained from reliable lines of reference. However, under the microscope the newly formed bone appeared to be easily distinguishable from the rest of the alveolar process, both in staining quality and in structural pattern (Fig. 2 A). From Table II it appears that the interval of time elapsed since the extraction of the antagonists and the variation in occlusal stress influenced the amount of bone apposition to a greater extent than the age of the patient. It was remarkable that the cervical 3-4 millimeters of the labial alveolar process was completely rebuilt over a period of 169 days in a patient 64 years old (case number 14). This histologic observation confirms the clinical impression and experience that orthodontic movement of teeth can be accomplished with a satisfactory result in relatively old individuals.

The rapid apposition of alveolar bone in several of these experimental cases obviously necessitated a reorganization of the attachment of the periodontal fibers. Relatively widely separated heavy coarse fiber bundles were observed to enter the alveolar bone with one to five large cuboidal osteoblasts lined up between the individual fiber bundles. The attachment of the periodontal fibers to the cementum appeared unaltered. Very little cementoid or new cementum could be observed on the root surfaces except in areas of repair following previous traumatic injuries.

The gingival and alveolar crest fibers had maintained a distinct functional orientation during the limited time (up to 169 days) that has elapsed following the loss of the oppposing teeth. Several areas of well defined oblique fibers also could be observed in the periodontal membrane, but more often the fibers were oriented parallel to the surface of the root and the alveolar bone. Invariably the thin spindle shaped older connective tissue cells were lined up in a typical functional arrangement, while the young fibroblasts had a non-functional arrangement. Inconspicuous changes of early degeneration and atrophy in some instances could be observed in the subcrestal fibers. Fragmentation and partial disintegration of the collagen fibers occasionally could be observed. This was seen best in sections stained with Azan connective tissue stains. In some instances the collagen fibers were replaced by a meshwork of reticulum-like fibers in a seemingly edematous environment representing areas of so-called "mucoid degeneration" (Fig. 8). A few plasma cells and lymphocytes commonly were

Fig. 12A. Compression and slight hyalinization of the periodontal membrane at the alveolar crest. Width of periodontal space: 0.10 mm. Note blood vessels with thick endothelial lining. Case 15. Magnification X250.

Fig. 12B. Clinical picture of case 15. The maxillary right lateral incisor was the experimental tooth. The occlusal stress on this tooth was heavy following extraction of the posterior teeth.

found within the periodontal membrane in areas of beginning disuse atrophy and degeneration. In none of these instances did the atrophy and degeneration approach in severity the changes of disuse atrophy that have been found in autopsy material when teeth have been without functional contacts for several years. Therefore, it can be assumed that disuse atrophy of the periodontal membrane progresses slowly in adult individuals. The relative stability of the collagen fibers in the fully developed adult periodontium also has been borne out in animal research on nutritional deficiencies[5].

No indication could be found of a so-called "intermediate plexus" of the periodontal membrane (Sicher [19], Orban[16]). Blood vessels and surrounding interstitial tissues commonly were located in the middle portion of the periodontal membrane, and it is conceivable that repair of the periodontal membrane following injury may originate in these tissues; but whenever functionally oriented periodontal fibers were observed, they exhibited a distinct continuity in their pattern or orientation from the surface of the cementum to the surface of the alveolar bone. Reorganization of attachment of the fibers seemed to take place on the surface of the alveolar bone and at the alveolar crest in response to changes in functional stress on the teeth. The intricate interwoven pattern of the periodontal fibers is obvious when cross sections and longitudinal sections through the root of a tooth and its surroundings are studied simultaneously. It is therefore virtually impossible to follow the entire length of a fiber bundle in the plane of any one section prepared for microscopic study. But the general pattern of orientation of the collagen fibers at the alveolar crest, the horizontal fibers, and the oblique periodontal fibers in this material did not indicate any seam or structural variation in the middle portion of the periodontal membrane. Neither were the early degenerative changes consistently localized to the middle portion of the periodontal membrane.

The most stable part of the periodontal membrane appeared to be the attachment to the cementum and the immediate vicinity of this attachment. Repair of the periodontal structures following trauma from occlusion is well illustrated in case number 3 (Fig. 3 C). The experimental tooth had been in traumatic occlusion up to 15 days prior to the removal of the specimen when the opposing teeth were extracted. Rapidly progressing repair of alve-

olar bone and cementum could be observed in this case.

The once case of a tooth from the mandible (case number 1) without antagonist followed the pattern of changes in the periodontal membrane already described for the eight cases from the maxilla, but there was no evidence of resorption on the labial aspect of the alveolar process. One would expect that the lower anterior teeth would move labially following loss of antagonist in contrast to the maxillary teeth that moved lingually. The lack of remodeling resorption on the labial aspect of the alveolar process in this case therefore can be understood. The lack of labial resorption furthermore was of great interest since a flap operation had been performed 14 days prior to the removal of the specimen. The lack of resorption following the flap operation in this case helps to exclude the surgical trauma as the cause of resorption on the labial aspect of the alveolar process in the previously described eight cases in the maxilla.

As could be expected, the periodontal findings in the six cases with increased occlusal stress varied considerably from the nine cases without antagonists. A process of remodeling of the alveolar process was observed also in the cases of increased occlusal stress, but here the remodeling represented an attempt to compensate for a labial movement of the teeth subsequent to the increased labial component of the occlusal stress. Apposition of bone was found on the labial aspect of the alveolar ridge and along the labial surface of the alveolar process. On the periodontal membrane side of the alveolar bone there was evidence of alternating areas of resorption and repair with the resorption dinstinctly dominating over the new bone formation. In four cases the labial bone plate was thick and made up of almost solid bone, but in two cases the labial alveolar process was perforated apically to the alveolar crest (Fig. 9 B). These perforations might have been caused by labial movement of the teeth. A similar perforation of the alveolar process also was found in case number 3 where the experimental tooth had been in traumatic occlusion until two weeks prior to the removal of the specimen.

Some areas of root resorption, occasionally extending into the dentin, were found associated with heavy occlusal stress. In some instances there was a marked tendency toward root resorption (Fig. 5 A), while in most instances (Figs. 10 A and 12 A) the root did not show any reaction to compression of the periodontal membrane. The zone of cementoid and newly formed cementum appeared slightly thicker on the teeth that had been subjected to heavy occlusion than on the previously discussed teeth with decreased occlusal function. But the cementoblasts appeared distinctly squamous in all instances and never were any cementoblasts observed that even approached the cuboidal appearance of the osteoblasts on the surface of the alveolar bone.

Normal functional arrangement of the periodontal fibers seldom was found in these cases with increased pressure from the occlusal forces. In some cases the periodontal space appeared widened; in other cases a definite compression of the periodontal membrane was evident at the cervical area with the fibers arranged parallel to the surface of the tooth and the alveolar bone. A beginning hyalinization at the middle portion of the periodontal membrane (Figs. 9 D and 12 A) was observed associated with the compression. As described by Oppenheim[12], dilatation of vessels (Fig. 12 A) could be found adjacent to the areas of hyalinization. In other areas vascular granulation tissue was observed in association with bone resorption (Fig. 4 B).

In areas where the labial bone plate was missing, the periodontal membrane assumed the morphological characteristics of periosteum (Figs. 9 B and 9 C). Attachments of muscle fibers into this area very closely resembled muscle attachment to regular periosteum. The osteoblasts associated with periosteum in any area were squamous and never approached the size and cuboidal

appearance of the osteoblasts that were seen on the surface of the alveolar bone. It may be assumed that the large size and cuboidal shape of these latter osteoblasts is an indication of the rapid rate by which bone can be deposited on the surface of the alveolar bone.

It was also interesting and thought provoking to note how vulnerable the root surface is to resorption following compression of the periodontal membrane. The midportion of the periodontal membrane seems to be most vulnerable to increase in pressure as evident by the early hyalinization and vascular changes in this area. Azan stains for connective tissue also revealed an alteration in staining quality in these areas of hyalinization as described recently by Stahl, et al.[20]

The stability of the Sharpey's fibers inserted into the cementum and the persistence of the alveolar crest fibers in the presence of great changes in the other periodontal structures were the two most striking observations in this study. These structures also were found to be very resistant to changes associated with scurvy in an investigation by Waerhaug[22]. The biologic stability of these structures appear to be of great significance in prevention of downgrowth of the epithelial attachment and pocket formation both in relation to traumatic and systemic periodontal changes.

SUMMARY AND CONCLUSIONS

1. Periodontal changes associated with functional decrease and increase in occlusal stress on 15 human teeth have been described.

2. The most stable periodontal structure with regard to functional changes in occlusal stress appear to be the Sharpey's fibers entering the cementum and the periodontal fibers coronally to the margin of the alveolar crest.

3. Most of the adaptation of the periodontal fibers to change in functional demand seemingly takes place at the surface of the alveolar bone and the middle zone of the periodontal membrane.

4. Disuse atrophy of functionally oriented periodontal fibers in adults is a slow process.

5. Loss of posterior teeth may lead to traumatic occlusion with such sequellae as resorption of the surface of the root extending into dentin, and resorption of the alveolar bone with perforation of the labial wall of the alveolar process.

6. Remodeling and partial rebuilding of the alveolar process in response to changes in functional occlusal stress occurred in all of the patients regardless of the variation in age from 16 to 68 years of age.

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Periodontosis—Associated with Gingival Lesions—in a Child

by R. A. CAWSON, M.B.B.S., B.D.S., F.D.S., R.C.S.,* *London, S.E.5, England*

THE following case is described rather for the sake of its peculiar features than for any light that it sheds upon disorders of the gingivae or of the supporting tissues of the teeth. The striking features of the case are the multiple discrete lesions of the gingival margins and the areas of gross resorption of alveolar bone without appreciable pocketing or suppuration.

History: The patient, a girl of 11 years, complained of painful ulcers in the mouth of about one week's duration. The ulcers had occurred at irregular intervals for two years, and each ulcer lasted about a week before healing. Nothing was known that precipitated the disorder and there was no history of ulcers in any other sites than the mouth. There was no complaint of any other symptoms.

Examination: The patient was rather pale. She was of normal development for her age and showed no obvious abnormalities. Within the mouth there was a healing aphtha on the buccal mucous membrane, and this was the source of the complaint. Much more conspicuous than this, however, were the lesions on the gingivae.

Gingivae and Mucous Membrane: The mucous membrane was of normal colour, but there was a mild chronic gingivitis with hyperplasia of the gingival margins especially in the upper and lower incisor regions.

Distributed along the gingival margins were nine discrete lesions: five were on the upper gingivae and four on the lower. The lesions were rounded in outline, varied from about 0.5 cm. to 0.75 cm. across and extended from the gingival margin towards the buccal sulcus (see Fig. 1). They were

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Fig. 1. One of the discrete vascular lesions in relation to the upper right central incisor.

bright red in colour and were sharply defined from the surrounding mucous membrane. The surface of the lesions was slightly raised, there was no blanching on pressure and there was no pain or tenderness.

Neither the patient nor her mother were aware of the lesions or of how long they had been present.

The Supporting Tissues: In the upper and lower incisor region there was false pocketing of a degree compatible with the gingival hyperplasia. The incisor teeth and especially the lower incisors were, however, freely mobile with 1 to 2 mm. movement at the incisal edge. The upper central incisors appeared in addition, to have drifted labially, having inclined in this direction and separated in the process. The right upper central incisor had moved further than its companion and was slightly extruded. In the case of the upper first molars there was pocket formation extending along most of the length of the anterior buccal roots.

The rest of the teeth showed no pocket formation or loosening.

The Teeth: The teeth were well formed and showed no caries or hypoplastic defects.

Radiographic Examination: There was gross resorption of the alveolar bone in the upper and lower incisor and upper first molar regions. From half to two-thirds of the alveolar bone was lost in these regions (Fig. 2).

HISTOLOGICAL EXAMINATION

Section of two typical lesions in the upper incisor region and canine regions showed the abnormal tissue to be sharply demarcated from the surrounding mucosa. The lesion consisted of vascular connective tissue with a heavy chronic inflam-

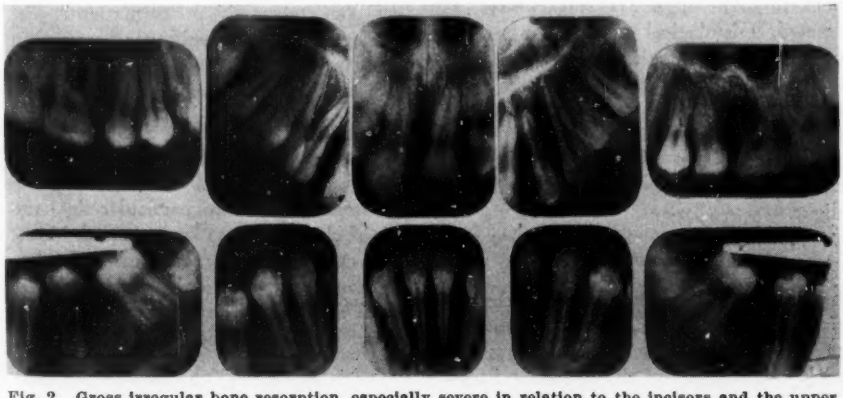

Fig. 2. Gross irregular bone resorption, especially severe in relation to the incisors and the upper first molars.

matory cellular exudate. The epithelium, which has no superficial layers, is ulcerated in places and shows considerable hyperplasia. Numerous blood vessels extend up through the epithelium. The marked inflammatory changes have probably masked the underlying pathology and the histological report concludes that "The appearances are non-specific but suggest that the lesion is a form of capillary haemangioma part of which has become infected and ulcerated". (Fig. 3.)

Fig. 3. Section of a biopsy of a gingival lesion showing the thinning of the epithelial covering, the numerous superficial blood vessels and the inflammatory exudate (X200).

GENERAL EXAMINATION

The patient showed no disorder of the skin. There was no increased keratinisation of the palms of the hands or the soles of the feet and no other obvious abnormalities.

Skeletal X-rays showed no abnormalities.

Blood examination showed Hb 116% and no abnormal features.

The child had not begun to menstruate and the urinary F.S.H. was less than 12 u in 24 hours.

No family history could be obtained of any disorders of the skin or oral cavity.

TREATMENT

In view of the chronic gingivitis, scaling of the teeth was done and the child instructed in how best to clean the teeth. Because of the bone destruction and pocketing, the upper first molars were extracted. Histological examination of the extracted teeth and the fragments of tissue which remained attached showed no angiomatous tissue. An attempt has also been made to improve the occlusion by fitting an Andresen-type appliance though the chances of success are, unfortunately, slight.

PROGRESS

During the nine months since the patient was first seen, the lesions on the mucous membrane have remained apparently unchanged and the periodontal condition has not deteriorated further.

DISCUSSION

The features of this condition that require explanation are, firstly the nature of the gingival lesions. Are they inflammatory, angiomatous, or a variety of "puberty gingivitis"? Secondly, what is the relationship, if any, of the gingival lesions to the alveolar bone loss?

The most important features of the gingival lesions are their vascular appearance, their sharply defined extent and their lack of progress either towards healing or spread. Further, the lesions cause no symptoms, are of unknown duration and may, therefore, be congenital malformations. The histology is of little assistance here owing to inflammatory changes which may have modified the original appearances to a considerable extent.

That the lesions are not simply inflammatory in origin is indicated by the clinical features and in particular by the lack of change over so long a period. Moreover, except in the region of the upper first molars, there is only a chronic gingivitis and these lesions are certainly not typical of that disorder.

The evidence, both clinical and biochemical is against the suggestion that the

mucosal lesions are due to endocrine influences.

There is no clinical evidence to show that the mucosal lesions are an example of Hyperkeratosis Palmo-plantaris, though this is one of the few conditions in which periodontosis may occur in a child (Jansen and Dekker, 1956; Rosenthal, 1951) or are part of any other skin disorder.

There remains, therefore, the probability that the lesions of the gingivae are a type of haemangioma, that is, a vascular malformation or naevus.

Haemangiomata of the capillary type occur in the mouth (Stones, 1957 and Thoma, 1954) but are uncommon. Multiple small haemangiomata on the oral mucous membrane do not appear to have been described and a positive diagnosis in this case is not justified.

Resorption of the bone underlying a vascular malformation such as a haemangioma is a well-recognised complication. It is tempting, therefore, to suggest that the gingival lesions are angiomata and are the cause of the alveolar resorption in the incisor and molar regions. Against such a suggestion, however, is the doubtful nature of the superficial lesions, and the fact that no bone resorption has occurred beneath the lesions in the premolar regions.

It is, however, too much to accept that the association of most of the gingival and bony lesions is merely fortuitous especially as these are both unusual disorders. Under the circumstances that no more satisfactory diagnosis can be made, therefore, it is suggested that this is a case of multiple oral haemangiomata causing alveolar bone resorption. In addition inflammatory changes have occurred in the angiomata and have caused suppuration and pocket formation around the upper molar teeth.

ACKNOWLEDGEMENTS

I am grateful to Dr. M. E. A. Powell of the Department of Morbid Anatomy for his report on the histology of this condition. My thanks are also due to Mr. W. Smith for the clinical photographs.

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DR. EDGAR D. COOLIDGE HONORED

Dr. Edgar D. Coolidge of Evanston, Illinois was presented recently with a 1958 Distinguished Service Award of the Illinois Interprofessional Council. This award is presented annually by the Council which is composed of members of the dental, chiropody, optometry, pharmaceutical and veterinary professions in Illinois. Dr. Coolidge was cited for his more than 52 years of "dedicated efforts and contributions to the health and welfare" of the people of Illinois.

Dr. Coolidge is professor emeritus of dental therapeutics at Loyola University School of Dentistry and author of several textbooks and scientific articles. He is also widely respected for his calm, mature judgment and idealistic viewpoint.

We are pleased that the Illinois Interprofessional Council saw fit to honor Dr. Coolidge.

The Effect of Physiologic and Pathologic Processes Upon Certain Histochemically Detectable Substances in the Gingiva*,**

by SAMUEL TURESKY, IRVING GLICKMAN, BEVERLY FISHER, *Boston, Mass.*

VARIOUS chemical groups and compounds have been demonstrated in gingiva by chemical and histochemical methods.[1-9] Although these substances have been identified with a wide range of physiologic processes (keratinization, enzymatic and antibody reactions, cell permeability, detoxification, biosynthesis, and chelation,[10-13]) and pathological changes in tissues other than the gingiva,[14,15] their significance in the gingiva remains to be determined. The present study was undertaken to determine (a) the distribution of protein-bound sulfhydryl, disulfide and amino groups, ribonucleic acid, glycogen and other polysaccharides in normal gingiva, (b) the modifications these substances undergo in the course of physiologic processes such as aging and keratinization, (c) the manner in which these substances are affected by inflammation.

PROCEDURE

148 specimens of normal and inflamed gingiva were obtained from 74 human subjects, (39 males, 35 females) from 10 to 81 years of age. Two contiguous rectangular shaped specimens were taken from each patient on the buccal surface of the maxillary or mandibular premolar and molar areas so as to include the marginal and attached gingiva, and the alveolar mucosa (Fig. 1). One specimen was fixed in a one per cent solution of trichloracetic acid in 80% alcohol for a period between 18-24 hours, and the other was fixed in a neutral alcohol-formalin solution (10% formalin in 95% alcohol buffered to pH 7.0 ± 0.3) for the same period of time.

Seven slides in serial section were prepared from each of the specimens as follows: slide 1, stained with hematoxylin and eosin for purposes of orientation and morphologic study; slides 2 and 3, prepared according to the Barrnett-Seligman technic for the demonstration of protein-bound sulfhydryl and disulfide groups;[16] slide 4, prepared according to the Weiss, Tsou, Seligman technic for the demonstration of protein-bound alpha amino groups;[17] slides 5, 6, 7 prepared according to a modified Lillie Periodic Acid Schiff (P.A.S.) technic for the demonstration of glycogen and other polysaccharides;[2] slide 8, prepared according to the Methyl Green Pyronin Y method for the distribution of ribonucleic acid.[18]

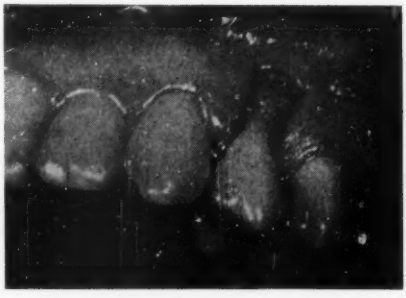

Fig. 1. Rectangular biopsy of marginal and attached gingiva and portion of alveolar mucosa.

MICROSCOPIC AND HISTOCHEMICAL OBSERVATIONS

Uninflamed Attached Gingiva: Each specimen consisted of marginal and attached gingiva and alveolar mucosa (Fig. 2). All specimens, even those which appeared normal clinically, presented some microscopic signs of inflammation in the marginal gingiva. This varied from a slight

*This report is from an investigation carried out under Contract #DA 49-007-MD 706 from the Department of the Army, Surgeon General's Office.

**From the Ziskin Memorial Research Laboratory, Department of Oral Pathology and Periodontology, Tufts University School of Dental Medicine.

Fig. 2. Typical gingival biopsy of marginal and attached gingiva and portion of alveolar mucosa. Hematoxylin and eosin (orig. mag. x 28).

cellular infiltration to a pronounced accumulation with increased vascularity and degenerative and proliferative changes in the cervicular epithelium and connective tissue. In cases of severe marginal involvement, the inflammation extended into the attached gingiva. In order to study the histochemical features of the gingiva without the complicating effect of inflammation, observations were confined to uninflamed attached gingiva. Because inflammation was present in the attached gingiva in all specimens beyond the age of 50, these specimens were not considered in determining the features of normal gingiva.

Morphologic Pattern of the Epithelium: Because of the variation in the configuration of the epithelium of the attached gingiva from specimen to specimen, it was necessary to use a standard for purposes of comparison. The method selected was that of Wentz et al.,[19] with modifications. The epithelium of the attached gingiva of each specimen was classified according to measurements of ridge length and suprapapillary width made with a Bausch and Lomb micrometer disc, as follows:

1. Epithelial ridge length (L) was defined as the distance from the epithelial surface to the tip of the ridge (Fig. 3).

2. Suprapapillary width (W) was defined as the distance from the surface to the base of the epithelium at the tip of the connective tissue papilla (Fig. 3).

Averages were computed of the three longest epithelial ridges and the narrower of the adjacent suprapapillary zones. Tangentially cut sections were not used.

3. $\frac{\text{The ratio of epithelial ridge length (L)}}{\text{suprapapillary width (W)}}$

was used as the index of the extent of epithelial projection into the underlying connective tissue.

The following values were obtained for uninflamed attached gingiva:

Average epithelial ridge length (L) . . . . 335 mu ± 50 mu, with a range of from 190 to 500 mu. Average suprapapillary width (W) . . . . 105 mu ± 25 mu, with a range of from 55 to 185 mu. Average $\frac{L}{W}$ ratio . . . . 3.4 ± 0.8, with a range of from 1.5 to 4.9.

Relation of Histochemical Findings to Morphologic Pattern of the Epithelium: Glycogen in varied amounts was found in the epithelium of the uninflamed attached gingiva in 30% of the patients. It appeared in the prickle cell layers and tended to

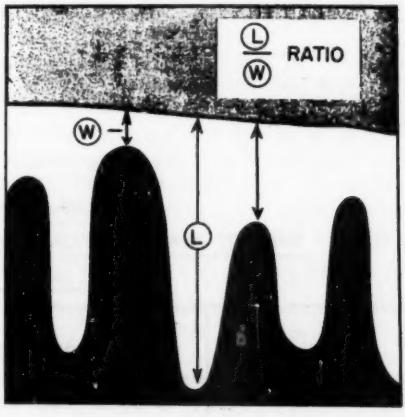

Fig. 3

Fig. 3. $\frac{L}{W}$ ratio used for study of epithelial configuration in attached gingiva. L is the distance from the surface of the epithelium to the tip of the ridge. W is the distance from the surface to the base of the epithelium, at the tip of the connective tissue papilla.

occur in the specimens with the largest $\frac{L}{W}$ ratio values (Table 1).

In all specimens of uninflamed attached gingiva the surfaces were either keratinized or parakeratinized. Both types of surface structure often occurred in the same specimen. There appeared to be an inverse relationship between the presence of surface keratinization and the occurrence of glycogen. Glycogen was found in the epithelium in only 8% of the cases in areas in which the surface was keratinized, compared with 26% of the parakeratinized areas (Table II). When present, the glycogen was found in the prickle cell layer.

The distribution of protein-bound sulfhydryl and disulfide groups, ribonucleic acid, glycogen and other P.A.S. positive substances in the gingiva described in previous reports[2,3,4,6] was corroborated by our findings. Protein-bound amino groups, not previously reported in the gingiva were observed in both the epithelium and connective tissue. Throughout the epithelium they stained dark blue in the nuclei and light purple in the nuclear membrane, cytoplasm and cell membrane. These groups appeared in greater concentration in the surface keratinized and parakeratinized layers than in the remainder of the epithelium (Fig. 4). Variations in the morphologic pattern of the uninflamed epithelium were not accompanied by differences in the distribution of protein-bound sulfhydryl, disulfide or amino groups or ribonucleic acid.

Diastase-resistant polysaccharides (P.A.S. positive substances other than glycogen) were present in the cell membranes

Table I

UNINFLAMED ATTACHED GINGIVA
THE PRESENCE OF GLYCOGEN AND
MORPHOLOGY OF EPITHELIUM

SUBJECTS	$\frac{L}{W}$	SUBJECTS SHOWING GLYCOGEN IN EPITHELIUM	%
3	1-1.9	0	0
9	2.0-2.9	1	11
20	3.0-3.9	7	35
8	4.0+	4	50

Table II

UNINFLAMED ATTACHED GINGIVA
INVERSE RELATIONSHIP BETWEEN
SURFACE KERATINIZATION AND THE OCCURRENCE OF GLYCOGEN
IN THE EPITHELIUM

	AGE (40 cases)		SEX (40 cases)		PRESENCE OF GLYCOGEN
	10-29 (16 cases)	30-49 (24 cases)	M (18 cases)	F (22 cases)	40 cases
KERATINIZED	38%	73%	56%	59%	8%
PARAKERATINIZED	63%	36%	50%	50%	26%

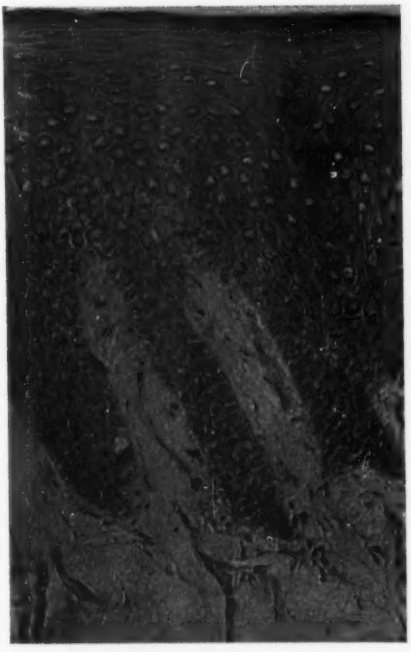

Fig. 4. Uninflamed attached gingiva, showing distribution of protein-bound amino groups in the epithelium and connective tissue. In the epithelium they are present in the nuclei, nuclear membrane, cytoplasm and cell membrane. Note the greater concentration in the surface parakeratinized layer. In the connective tissue, the protein-bound amino groups are present in the fibroblasts and endothelial cells. Weiss, Tsou, Seligman technic (orig. mag. x 420).

throughout the epithelium and appeared more prominent as the surface was approached and the cells became flattened. There was an inverse relationship between the stainability of these substances and the degree of keratinization. Comparison of keratinized attached gingiva (Figs. 5 and 6) with adjacent parakeratinized alveolar mucosa (Figs. 7 and 8) demonstrated this inverse relationship between the occurrence of diastase-resistant polysaccharides and keratinization.

Connective Tissue: In the connective tissue of the uninflamed attached gingiva, protein-bound sulfhydryl, disulfide, and amino groups and ribonucleic acid were present in the fibroblasts, endothelial cells, and smooth muscle cells of the blood vessels. Only disulfide groups in small amounts, and sulfhydryl groups in lesser amounts were present in the intercellular substance.

Glycogen appeared homogeneously throughout the intercellular substance of the connective tissue, and in the smooth muscle of arterioles. P.A.S. positive substances, other than glycogen, were present in the basement membrane, in the intercellular substance and the cell membrane of the fibroblasts. In the arterioles these substances were seen in the cell membrane of the endothelium and smooth muscle cells and in the connective tissue surrounding the internal elastic membrane.

THE EFFECT OF AGE AND SEX UPON THE GINGIVA

To determine the effects of age and sex upon the gingiva, the specimens in which the attached gingiva was free of inflammation were divided into two age groups, 10-29 years and 30-49 years. Specimens beyond the age of 50 were not considered because inflammation, present in the attached gingiva in all cases in this age group, complicated the microscopic picture.

Increased values obtained for the epithelial ridge length and suprapapillary width in the attached gingiva suggested that the epithelium was thicker in the 30-49 year group, but this finding was not statistically significant. Surface keratinization was observed twice as often in the 30-49 year group as compared with the 10-29 year old patients (Table II).

Glycogen was present in the epithelium of 25% (6 out of 24 cases) in the older group as compared with 50% (8 out of 16 cases) in the younger group. The walls of the arterioles in the older group stained more prominently for diastase-resistant glyco-protein than in the younger group. The distribution of protein-bound sulfhydryls, disulfides and amino groups and ribonucleic acid was was not affected by age, up to age 50.

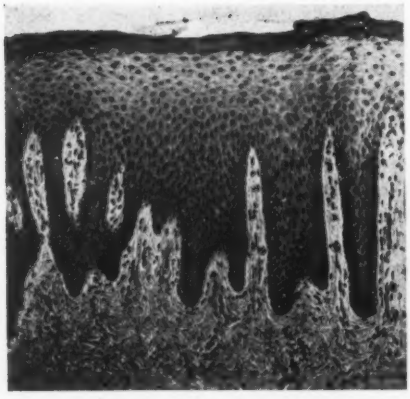

Fig. 5. Uninflamed attached gingiva, showing keratinized surface. Hematoxylin and eosin (orig. mag. x 200).

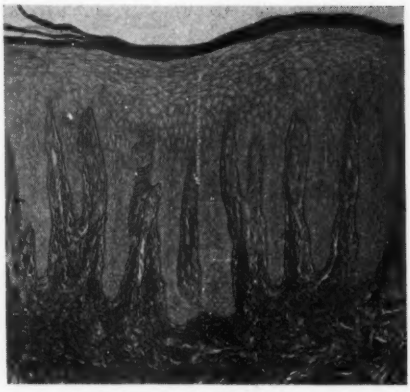

Fig. 6. Serial section of specimen shown in Fig. 5. Uninflamed attached gingiva, showing distribution of P.A.S. positive polysaccharides other than glycogen. Note the deeper staining of the cell membranes in the epithelium as the keratinized surface is approached. The basement membrane, ground substance and collagen fibers can be seen in the connective tissue. Periodic-acid-Schiff technic (orig. mag. x 200).

No sex difference was observed in epithelial configuration, occurrence of keratinization or the distribution of the various chemical groups and substances.

THE EFFECT OF INFLAMMATION UPON THE GINGIVA

As noted above, there was microscopic evidence of some inflammation in the marginal gingiva of all specimens. In cases of severe marginal involvement, the inflammation extended into the attached gingiva. In the patients, aged 50 and beyond, inflammation was present in the attached gingiva in all cases. In all specimens, degenerative changes secondary to inflammation were observed more frequently in the marginal gingiva because of the more pronounced involvement in this area.

In the inflamed attached gingiva the principal finding in the epithelium was proliferation which resulted in elongation and broadening of the epithelial ridges. Degeneration of epithelium was occasionally seen but was confined to individual cells or isolated groups. The maximum epithelial ridge length was 600 mu as compared with 500 mu in the uninflamed attached gingiva. Narrowing of the suprapapillary width accompanied the elongation of the epithelial ridges in the inflamed areas. The narrowest suprapapillary width in the inflamed attached gingiva was 25 mu as compared with 55 mu in the uninflamed gingiva. The largest $\frac{L}{W}$ value was 15 as compared with 4.9 in the uninflamed gingiva. Keratinization occurred infrequently in the inflamed attached gingiva at all ages (18% of the 34 inflamed specimens as compared with 55% of the 40 non-inflamed specimens).

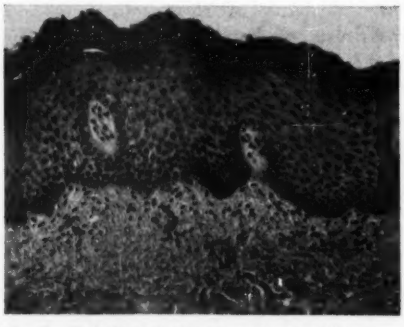

Fig. 7. Uninflamed alveolar mucosa adjacent to the attached gingiva shown in Fig. 5. Note the parakeratinized surface. Hematoxylin and eosin (orig. mag. x 200).

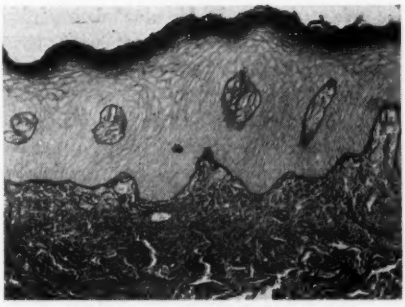

Fig. 8. Uninflamed alveolar mucosa, same area as Fig. 7, showing distribution of P.A.S. positive polysaccharides other than glycogen. Note that the parakeratinized surface stains more deeply than the keratinized surface layer of the adjacent attached gingiva shown in Fig. 6. Periodic-acid-Schiff technic (orig. mag. x 200).

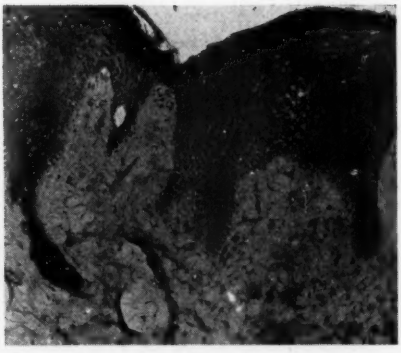

Fig. 10. Inflamed attached gingiva, showing accumulation of protein-bound amino groups, particularly prominent in the tip of elongated epithelial ridge. Compare with Fig. 4. Weiss, Tsou, Seligman technic (orig. mag. x 370).

Glycogen was present in the epithelium in 94% of the specimens of inflamed attached gingiva as compared with 30% in the absence of inflammation. In both the inflamed and uninflamed specimens the glycogen was prominent in the core of the elongated epithelial ridges (Fig. 9). Protein-bound sulfhydryls, amino groups and ribonucleic acid stained more densely in the epithelium of the inflamed attached gingiva and appeared to be related to the elongation of the epithelial ridges (Fig. 10). Associated with the fluid and cellular exudate of inflammation there was a thinning and diminished staining of the basement membrane and diminished staining of the connective tissue ground substance with the P.A.S. technic (Fig. 9).

Fig. 9. Inflamed attached gingiva, showing elongated epithelial ridge, with glycogen in the suprapapillary zone and in the core. At the tip of the ridge the basement membrane is thinned and lighter staining. Periodic-acid-Schiff technic (orig. mag. x 340).

DISCUSSION

Previous descriptions of histochemical features of the gingiva have been difficult to interpret because they dealt with the marginal area which was invariably inflamed. This introduced the problem of differentiating between the histochemical characteristics associated with physiologic processes and those introduced by inflammation. The present study, by concentrating upon the attached rather than the marginal gingiva, provides a basis for evaluating the morphologic and histochemical features of the gingiva, without the complicating effects of inflammation as well as the modifications which occurred when inflammation was present.

The initial finding which is of interest is that there is considerable individual

variation in the average epithelial ridge length in the uninflamed attached gingiva. This variation was not accompanied by cellular changes or alterations in the shape of the epithelial ridges which could be interpreted as indicative of increased cellular activity.

All specimens of uninflamed attached gingiva presented either keratinization or parakeratinization or both types of surface structure.

Glycogen was rarely seen in the epithelium if the surface was keratinized. It occurred in approximately 30% of the specimens with a parakeratinized surface. When present, the glycogen was generally found in the prickle cell layer. The inverse relationship between the presence of glycogen and surface keratinization is not unique to the gingiva. It has also been reported in skin.[14] The role of glycogen in the normal morphogenesis from basal to surface epithelial layers is not understood. Our findings corroborate the inverse relationship between the occurrence of keratinization and glycogen but provide no basis for explaining its significance.

Also of interest is the fact that glycogen occurred more often in the uninflamed attached gingiva in the specimens with the longer epithelial ridges. It was invariably present, and in greater concentrations, in the hyperplastic epithelial ridges in inflamed attached gingiva. From the latter finding it might be assumed that the appearance of glycogen is in some way associated with proliferative cellular activity.

P.A.S. positive polysaccharides other than glycogen were present in the cell membranes throughout the epithelium but stained more deeply in the keratinized and parakeratinized surface layers. There was an inverse relationship between the stainability of these substances and the degree of keratinization. This is in accord with the observation of Wislocki et al.[20] in epithelium of skin and mucous membranes. These findings indicate a greater availability of water insoluble, alcohol insoluble polysaccharides or substances with 1, 2 glycol groups and their substituted forms in the epithelial surface. However, these substances are less available for staining in keratinized as compared with parakeratinized areas. This may indicate that in the transition from parakeratinized to keratinized structure the degree of polymerization of the polysaccharides is increased so that fewer 1, 2 glycol groups can be oxidized to Schiff stainable aldehydes. On the other hand, depolymerization of the polysaccharides to water or alcohol soluble aggregates may account for the diminished staining of the keratinized structures.

Our evaluation of the effect of aging and sex difference upon the gingiva under physiologic conditions was based upon specimens from patients below the age of 50. This was necessary because inflammation was present in the attached gingiva in all specimens beyond this age group. There was an increased frequency of occurrence of surface keratinization and a less frequent appearance of glycogen in the epithelium of the 30-49 year group as compared with the 10-29 year old patients. In the connective tissue, diastase-resistant polysaccharides stained more intensely in the walls of the arterioles in the older group. This was associated with a thickening of the arteriolar wall. No differences were observed in other features of the connective tissue such as density of ground substance and fibroblast collagen ratio at the various age levels in this study.

There were no sex differences in the histologic or histochemical features of the epithelium or connective tissue.

The effect of inflammation upon the gingiva varied with its severity. The degenerative changes which were common in the marginal area were not present in any appreciable degree in the attached zone. Proliferation of the epithelium accompanied by thinning of the basement membrane and diminished staining of the connective tissue ground substance were the

principal changes introduced by the inflammation. Protein-bound sulfhydryl and amino groups and ribonucleic acid, normal components of the epithelium, were increased in hyperplastic ridges. Surface keratinization was observed less frequently where inflammation was present.

SUMMARY AND CONCLUSIONS

Microscopic and histochemical features of uninflamed and inflamed marginal and attached human gingiva were studied in 148 specimens obtained from 74 male and female subjects from 10 to 81 years of age.

There was an inverse relationship between the occurrence of glycogen in the epithelium and surface keratinization. Keratinized structure contained less P.A.S. positive polysaccharide than parakeratinized structure.

With age, the occurrence of keratinization was increased and the presence of glycogen in the epithelium decreased.

There were no sex differences in the gingival epithelium or connective tissue.

Glycogen, protein-bound sulfhydryls and amino groups and ribonucleic acid were increased in the epithelium in areas of hyperplasia associated with inflammation.

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The Effects on the Epithelial Attachment of Extraction of Adjacent Teeth in Macacus Rhesus Monkeys

by H. E. SIMPSON, M.D.S., *Newcastle upon Tyne, England*

WHEN it is necessary to extract a tooth the suspicion often arises that the epithelial attachment of the adjacent teeth will be damaged. The problem is similar whether it is believed that the attachment is organic or that it is purely mechanical, although in the former case any injury would be more serious since it would involve the breaking down of tissue as opposed to the separation of ununited structures.

In an attempt to throw some light on this problem, 14 extraction wounds in monkeys were examined with respect to the epithelial attachments of the teeth bordering the sockets. The extracted teeth had been removed, with as little trauma as possible, at intervals up to eight weeks before the conclusion of the experiment.

EXPERIMENTAL MATERIAL

The earliest specimens were 3 days old. Three of these were examined and in two of them there was evidence of injury extending beyond the area of the socket. Both were mandibular second premolar sockets. Fig. 1 shows the epithelial attachment on the distal side of a first premolar following the extraction of the second premolar. Part of the epithelium (A) was probably in contact with the enamel of the tooth and the remainder (B) with the cementum. The slight separation from the cementum may be an artifact.

The socket is filled with blood clot part of which is shown (E). Broken transeptal fibres of the periodontal membrane (F) are attached to the tooth and extend horizontally towards the socket.

A tear has occurred in the interdental papilla extending from the socket to the base of the epithelial attachment. It is not clearly seen because of the numerous red and white cells which are present but its position is indicated by the epithelial cells (D) which have proliferated along it towards the tooth. The most interesting feature, however, is the proliferation of cells from the lowest part of the epithelial attachment which are also growing along the line of the defect (C).

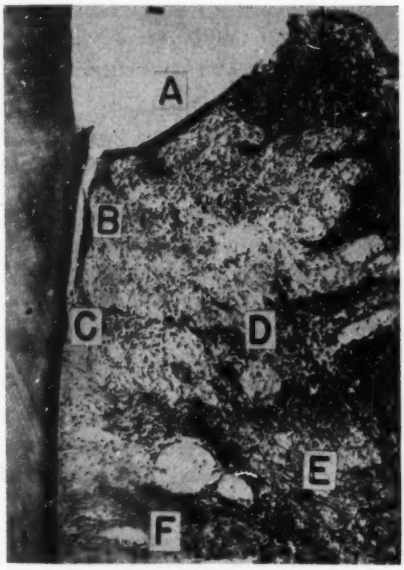

Fig. 1. Epithelial attachment on distal aspect of a mandibular first premolar 3 days after the extraction of the second premolar. For description see text. X100.

Fig. 2 shows the epithelial attachment on the mesial surface of a mandibular first molar in similar circumstances. In this case the whole of the epithelial attachment has been situated on the cementum. The attachment is divisible into two parts. From point (A) upwards the epithelium has probably been in contact with the cementum without any adhesion but from (A)

Sutherland Dental School.

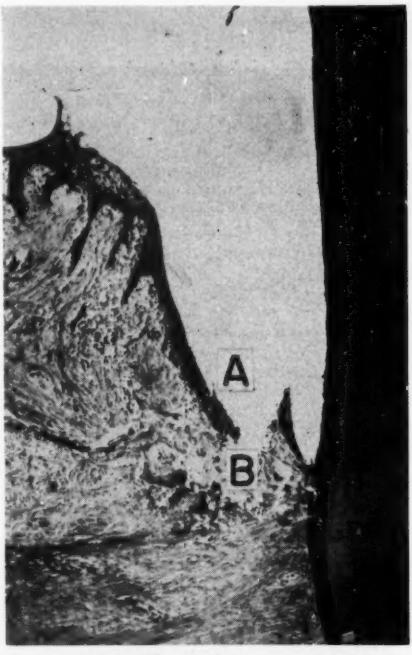

Fig. 2. Epithelial attachment on mesial aspect of a mandibular first molar 3 days after the extraction of the second premolar. For description see text. X100.

downwards, a closer connection is indicated by the presence of torn off cells adherent to the surface of the cementum.

It appears that the tearing away of the epithelium from the cementum has occurred during the extraction of the adjacent tooth. The main reason for this supposition is the small tear in epithelium and connective tissue at (B). The cells in the space formed by this tear are red blood cells which indicates that the injury occurred during life. The absence of a marked inflammatory reaction would be accounted for by the return to position of the epithelium following the extraction so that infection was excluded. The present separation is probably artifact consequent on the lack of adhesion of the previously separated epithelium to the cementum. Examination of further sections suggests that the tear may extend as far as the socket but it is not possible to demonstrate this with certainty.

In the remaining specimens it was not possible to find any definite evidence of trauma. The frequent presence of calculus meant that some degree of disturbance of the epithelium could normally be expected and a small injury can be sufficiently repaired in a week to render it unrecognizable in the presence of a coincidental inflammatory state. Occasional prolongations of epithelium from the socket towards the interdental crest suggested that tears of the type shown in Fig. 1 are not rare but none could be traced to the adjacent tooth.

Two generalizations can be made concerning the older specimens. First, although the height of the epithelial attachment varies on different parts of the same tooth, it tends to be lower on the side adjacent to a socket. Secondly, the numbers of inflammatory cells in the area of the attachment is usually greater on the socket side.

SUMMARY AND CONCLUSIONS

Fourteen extraction wounds in macacus rhesus monkeys were examined in an attempt to discover if any injury had occurred to the epithelial attachment of teeth adjacent to the sockets.

In the earlier specimens there was evidence that an injury could occur in the form of a tear which might extend across the interdental septum from the socket to the tooth.

In the later specimens, one week old and upwards, there was little evidence of injury although it was observed that the level of the attachment tended to be lower and that there was generally a more marked infiltration of inflammatory cells below the epithelium on the side of a tooth adjacent to a socket.

The Surface of Cementum*

by LAMBERT A. BENSON, D.D.S., M.S.**, *Columbus, Ohio*

CEMENTUM is a bone-like tissue which covers the surface of the anatomic root of the tooth and serves several functions.[10] Knowledge of the unexposed cemental surface is an essential prerequisite to understanding its pathosis and aids in establishing rational therapy.

The object of this investigation was to study the surface of cementum which serves as a component of the functioning attachment apparatus. The periodontal membrane and cementoid was removed by a method which presented the apparent normal appearance of the cemental surface. It was found that variations of the surface were related to incremental growth, smooth and irregular surface formation.

There is a conspicuous paucity of information in the literature pertaining to the normal surface of cementum. Since its discovery in 1835,[1] the major investigations of cementum have been concerned with its formation, microscopic anatomy and function.

MATERIALS AND METHODS

A total of 109 human teeth with complete histories were taken at random for this study. The selection included both anterior and posterior teeth obtained from patients entering the dental clinic at the Ohio State University, College of Dentistry. In all specimens, the location of the epithelial attachment to cementum was noted.

Three methods were used to remove the periodontal membrane and the cementoid.

The first method consisted of removing the periodontal membrane and cementoid of 40 teeth by mechanical means. The instruments used were the Gracy and McCall curettes, Ball sickle scalers, Skinner and Bunting files, and the ultrasonic scalers designed by Johnson and Wilson.[4]

Ethylenediamine, an organic reagent had been reported to have great penetrating ability.[6, 11, 18] After extraction twenty teeth were washed in cold water and any excessive gingival tissue was removed with a scalpel being careful not to injure the surface of cementum. They were then placed in a Florence flask containing 100 ml. of aqueous 80 per cent ethylenediamine solution in which they were allowed to remain from two to eight hours at increased temperatures ranging from 120 to 460 C.

Enzymatic debridement with trypsin was the third and most successful method. After extraction 49 teeth were washed in cold water and any excessive gingival tissue was removed with a scalpel as in the previous method. The teeth were then placed in a 4 ounce bottle sprinkled with approximately 2 grams of trypsin and diluted with a standard buffer solution of pH 7.0. Since 40 to 50 per cent of trypsin enzyme's proteolytic activity is dissipated within three hours,[8, 9, 14] the above procedure was repeated at the required intervals until the periodontal membrane and cementoid was digested. The extracted teeth in the buffered solution of trypsin was maintained as near as possible at 37 C. to maintain the maximum effectiveness of the enzyme.[9, 14]

After removal of organic material from the clinical root surface by the above methods, all specimens were examined under a dissecting microscope and an "Ultropak" microscope with relief condensers.* Photographs were made with low angle illumination at various magnifications.

RESULTS

A typical root surface of an extracted tooth showing the attached portions of the severed periodontal membrane covering the surface of cementum is shown in Figs. 1 and 2. It is obvious from these photographs that it is impossible to study the

*This paper is based on a thesis submitted in partial fulfillment of the requirement for the Master of Science Degree at the Ohio State University.

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The opinions or assertions contained in this article are the private ones of the writer and are not to be construed as official or reflecting the views of the Navy Department or the naval service at large.

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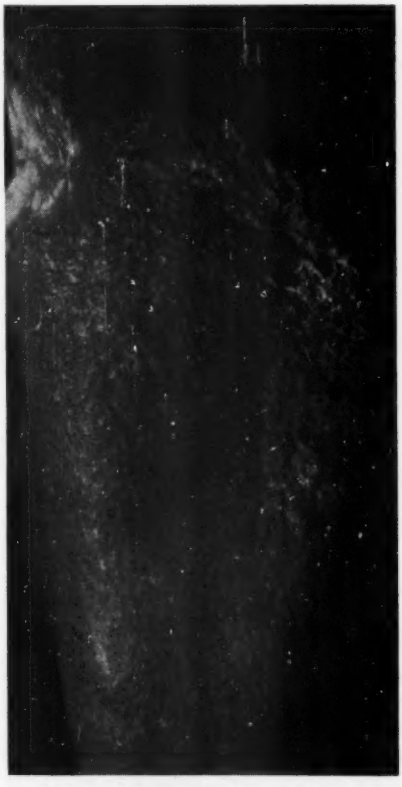

Fig. 1. Mesial surface of extracted lower right first bicuspid of a patient 68 years old showing the attached periodontal fibers. Orig. mag. 9X.

surface of cementum without removing the adherent periodontal membrane.

Hard nodular bodies were observed on approximately one-third of the teeth when studying the surface of roots before the removal of the organic tissue. These objects were attached cementicles (Fig. 2).

Microscopic examination of the cementum surface after mechanical removal of the periodontal membrane and cementoid revealed nicks, cuts and scratches reflecting the use of the instruments. This method was discarded because it was impossible to remove the organic material by mechanical means without altering the surface of cementum.

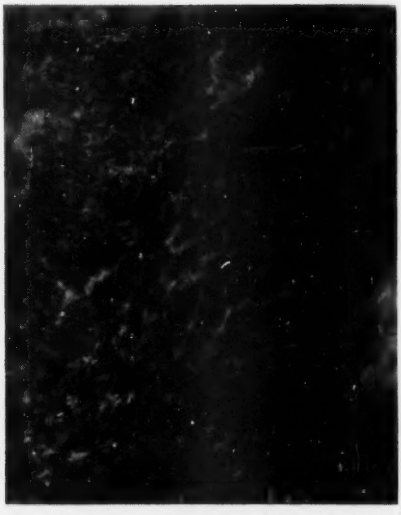

Fig. 2. Higher magnification Fig. 1. Arrow points to attached cementicle. Orig. mag. 22X.

Ethylenediamine had good penetrating value. It not only removed the organic tissue from the surface but also removed organic material below the surface of cementum. Following its use there were surface eminences and crater-like excavations (Fig. 3). The eminences fractured readily when tested with a dissecting needle and were found to be hollow. The resultant area resembled the above mentioned craters (Fig. 3). It was further observed that, during photography when the specimens were dry, the eminences fractured spontaneously resulting in formation of craters. On the basis of the above observations, the eminences and craters found on the surface of cementum after the treatment of ethylenediamine were deemed artifacts.

The eminences and craters appeared in all teeth treated with ethylenediamine. Sometimes they followed the imbrication lines of the surface of cementum as can be demonstrated in the coronal third of the root surface shown in Fig. 4. On other teeth there seemed to be no consistent appearance. One area of the root may be void of eminences whereas in an adjcent area they were concentrated (Fig. 3). The fact

Fig. 3. Mesial surface of mesial root lower right first molar after ethylenediamine treatment. Patient age 46. Line indicates epithelial attachment to cementum. Note crater-like excavations (c) and eminence (E). Orig. mag. 9X.

Fig. 4. Mesial surface of bicuspid after ethylenediamine treatment. Patient age 32. Line indicates epithelial attachment to cementum. Orig. mag. 9X.

that ethylenediamine produced artifacts on the surface of cementum rendered this method unsatisfactory for the purpose of this study.

The most desirable surface was obtained after digestion with trypsin. Trypsin lacked the penetrating power of ethylenediamine and digested only the organic material from the surface of cementum.

In general the surface appearance of cementum was that of smooth unglazed porcelain. The color varied from white to very light yellow. The surface was easily penetrated and scratched when probed with a dissecting needle. The topography of the root varied from a smooth, in fact almost polished, surface to one with convex knobby projections and crater-like depressions (Fig. 5). The smooth, glassy surface texture was found most frequently in the cervical third of the root. The irregularities were more prevalent in the apical third.

A summary of the significant surface appearances may be seen in Table I.

Of particular importance was the observation of imbrication lines in 35 per cent of the teeth studied after trypsin debridement. The imbrication lines are the shallow furrows encircling the circumference of the roots of the teeth. They are comparable to the perikymata of enamel except with wider intervals. It presented the concept of cones forming upon each other with the increments becoming progressively shorter toward the apex. These imbrication lines were more easily visible on teeth of younger individuals. In this study 13 of the 25 teeth from patients 40

TABLE I

Variety of Irregularities of Cemental Surface on Forty-nine Teeth After Debridement with Trypsin

	Age (yrs.) 10-25	*Age (yrs.) 25-40*	*Age (yrs.) over 40*	*Total*
Number of teeth	*7*	*18*	*24*	*49*
Irregularities				
Imbrication lines	4	9	4	17
Over all nodular appearance	0	0	6	6
Minute isolated nodules	1	3	9	13
Hypercementosis	3	2	4	9
Pitted or cratered	1	3	5	9

years or younger had visible imbrication lines. However, imbrication lines were observed on only 4 teeth of the 24 teeth from patients over 40 years. The furrows were more numerous and closer together in the cervical third of the root and were not observed close to the apex. (Fig. 6).

In addition to imbrication lines other irregularities were observed, such as nodules covering an entire surface. (Fig. 7 illustrates generalized nodules on the surface of a lingual root of an upper second molar. Cemental surfaces having an overall nodular appearance, as mentioned above, were observed in 12 per cent of the teeth studied after trypsin debridement. They resembled small blebs (Fig. 2) measuring approximately one-fourth of a millimeter in diameter. They were easily removed with a dissecting needle and appeared to have the same consistency as cementum. One specimen presented two similar larger nodules, one on the mesial the other on the distal. They were egg shaped and measured 1 mm. by 1½ mm. (Fig. 8).

In addition to the isolated minute nodules and more generalized nodules mentioned above, it was observed that 19 per cent of the roots of the teeth presented other forms of hypercementosis. The site of hyperdevelopment was usually in the area of the apex. The hyperdevelopment

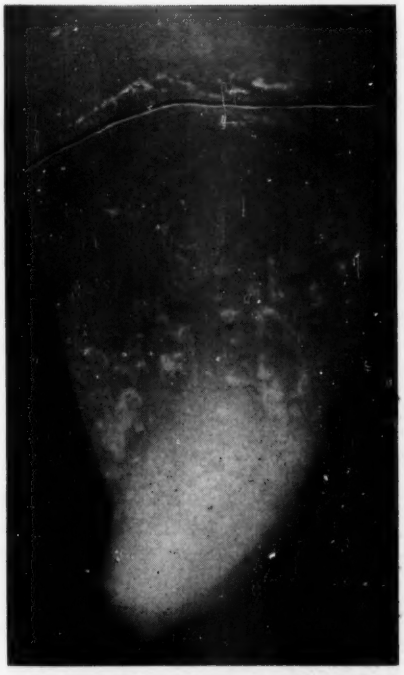

Fig. 5. Surface area of mesial root of 3rd molar after trypsin debridement. Patient age 23. Line indicates epithelial attachment. Orig. mag. 9X.

was not uniform but presented areas of greater and lesser thickness. (Fig. 5).

DISCUSSION

The development of a technic to debride the surface of cementum of organic material established a significant standard for the surface of the tissue. Rejection of mechanical methods, since they destroy the surface characteristics, bears a relationship to the effectiveness of removing calculus. This has been reported in the literature[6, 12, 15, 16] and the findings in this study corroborate the results of previous investigations. It is of particular significance to compare the results of mechanical removal of the attached soft tissue and its associated injury to the normal cementum surface. This aspect of the investigation emphasized the need for establishing a desirable surface of the tooth root after periodontal therapy.

Fig. 6. Mesial root second molar trypsin debridement. Patient age 32. Line indicates epithelial attachment to cementum. Note imbrication lines cervical ⅔ of the root. Orig. mag. 9X.

Fig. 7. Surface of lingual root upper second molar trypsin debridement. Patient age 63. Line indicates epithelial attachment to cementum. Note nodular appearance of the entire surface. Orig. mag. 9X.

As used in this experiment to remove organic tissue from the surface of cementum ethylenediamine is not effective. It has the unusual property of rapid and deep penetration. Observations of the results suggest that the affinity of ethylenediamine for the organic constituents of cementum is strong and that it extracts subsurface organic material readily. It is likely that ethylenediamine may have a more critical time of application than trypsin as an effective solvent for surface organic material and would require a special investigation to be adapted to this purpose. No effort was made to determine the cause of surface artifacts following attempted soft tissue removal with ethylenediamine. It may be postulated that the penetration of ethylenediamine is governed by distribution of organic material in cementum. If the organic material is not homogeneous the elevations and craters may be the result of the chemical reactions of proteolysis. It is also possible that the ethylenediamine penetrated to the granular layer of Tomes which is known to contain abundant organic material. Since the study was significantly related to the surface, and no histologic sections were made of the elevated areas, the problem remains unanswered (Fig. 3).

Trypsin, which digests the organic material, provided the most desirable result. As far as could be observed by the optical methods available in this study, the cementum surface remained undisturbed.

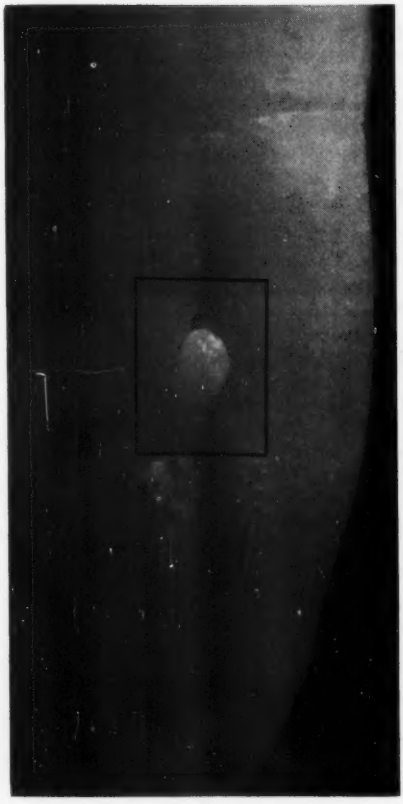

Fig. 8. Distal surface of root of upper 3rd molar trypsin debridement. Patient age 21. Note large nodule in boxed area. Orig. mag. 9X.

The cementum surface presented a variety of appearances. The first formed increments of cementum appeared to occur in the form of cones. The presence of imbrication lines indicated the subsequent cones failed to reach the cervix and terminated at a line apical to the previous increments. This verifies the accepted concept of cementum growth.[11,18] In the apical area the imbrication lines are not concentric, but the lamellae assume an indescribable irregular pattern. At times the irregular growth at the apex assumes the appearance of hypercementosis, which may be the result of the ever changing position of the teeth in their alveoli. The relatively smooth layer of cementum which was observed in 50 per cent of the specimens in the gingival "third" was not exposed in vivo. Careful notations of the epithelial attachment to cementum were made prior to the extraction of the teeth and the relatively smooth cervical area was a normal component of cementum with attached periodontal membrane.

In addition to lamellar growth of cementum, there appears to be a nodular formation in older teeth (Fig. 7). From this study no reason for nodular cementum formation was apparent. Nodular formation may cover the entire root or part of the root and seems to denote a change in function of periodontal membrane from the formation of the lamallae to one of isolated areas of cementum. The nodular development may be another form of denticle development deposited on the surface of existing cementum and is worthy of investigation.

The incidence of resorption recognized as Howships lacunae was so low it appeared to be insignificant. The use of mechanical methods and ethylenediamine prevented making accurate observations of resorption in specimens prepared by these methods. Of those teeth treated with trypsin only one identifiable area of resorption was recorded (Fig. 9). However, this is contrary to other investigations.[4,10] In Henry and Weinman's[4] investigation over 90 per cent of the teeth revealed histologic evidence of resorption of the root. It is possible that previously resorbed areas had been repaired. Since only the surface of the cementum was examined, these areas were not evident.

It is difficult to associate the attachment of calculus to the root surface with the reported irregularities of cementum. Of greater significance is the desire of the periodontist to produce a smooth surface and still retain the surface characteristics of cementum. While this study establishes the normal cemental surface, what constituted a desirable surface after removal of calculus is still to be established.

CONCLUSIONS

This investigation suggested a method of preparing the surface of cementum for examination. The cemental topography of a limited number of teeth was described. The results indicated the following significant observations.

1. The cementum may be adequately prepared for surface study by digesting the organic material with trypsin.

2. The use of ethylenediamine and mechanical methods resulted in the formation of artifacts.

3. The surface of unexposed cementum exhibited imbrication lines, isolated minute nodules, surfaces having an over-all nodular appearance and dish shaped areas. Hypercementosis manifested by irregular deposition occurred mainly in the apical area.

Grateful acknowledgment is made to Dr. John Wilson, Dr. William Lefkowitz and Dr. Duncan McConnell for their invaluable assistance in this study.

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Epidemiology and Indexing of Periodontal Disease*

by JOHN C. GREENE, D.M.D.,** *Washington, D. C.*

EPIDEMIOLOGICAL concepts had their beginning many years ago. More than 400 years before Christ, Thucydides wrote of his belief in observing facts and judging by rule of reason[1] and pointed to his observations relating to immunity during the plague of Athens.[2] At least one basic concept has persisted from this early beginning down to the present time—that is, the concept that epidemiology is concerned with groups of people. This concept is implied in the roots of the word which mean "on, upon, or to the people." Epidemiology's concern with groups of people is apparent in the following generally accepted definition of epidemiology: "This is the field of science which is concerned with the various factors and conditions which determine the occurrence and distribution of health, disease, defect, disability, and death among aggregations of individuals; it has application also to problems other than those of health and disease."[3]

Epidemiologic principles and methods currently are being applied not only to communicable diseases but also to other disorders "on, upon, or to the people" such as: heart disease; hypertension; mental disorders; nutritional deficiencies; accidents; congenital defects; mottled enamel; periodontal disease; and dental caries.

These disorders are a result of imbalance between a causative agent and man. The extent of imbalance depends on the characteristics of the agent and of man, both of which are in turn controlled by the environment. Thus, within the agent, the host, and the environment are all of the factors which affect the occurrence and progression of the disease or disorder. The combination of the processes which upset the balance between agent and host and initiate and perpetuate a disease is termed the natural history of the disease.

The body of knowledge about the natural history of any disease or disorder is made up of facts and observations concerning the agent, host, and environment which are assembled from many fields of science. They are combined with facts relating to the occurrence of disease according to age, race, sex, occupation, heredity, personal habits, and other host characteristics and facts concerning the physical, social, economic, and biologic environment. It is a function of descriptive epidemiology to collect and organize such data from many disciplines to describe the natural history of disease.

Epidemiologic description differs from clinical description in the unit of observation and in the type of information given. The epidemiologist describes characteristics of groups of people; the clinician describes the individual. The epidemiologist describes the factors related to the initiation and progression of the disease in the group; the clinician is concerned with the manifestations of the disease in the individual.

Epidemiology is not purely a descriptive science, but it is also a constructive science. Frost,[4] perhaps the greatest of American epidemiologists, stated that "Epidemiology is something more than the total of its established facts. It includes their orderly arrangement into chains of inference which extend more or less beyond the bounds of direct observation. Such of these chains as are well and truly laid, guide in investigation to the facts of the future. Those that are ill laid, fetter progress."

*Presented at the meeting of the American Academy of Periodontology, November 6, 1958, Dallas, Texas.

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Just what does the epidemiologist envision as his ultimate goal when describing or constructing the natural history of a disease in a group of people? What is the ultimate objective in studying the pathology or clinical behavior of disease? In broad terms, the answer to both of these questions is to gain a better understanding of the whole disease process in order to bring about its effective prevention and control. Since prevention depends on knowledge of the etiology, the principal goal in studying the epidemiology of a disease is to identify and determine the relative importance of all factors and conditions which may influence its occurrence and distribution. Therefore, investigators seek to acquire more knowledge about the whole chain of events surrounding a disease process with the anticipation that a vulnerable link in this chain will be exposed. Once a vulnerable link has been identified and practical methods of breaking this link are found, prevention becomes possible. Attainment of this goal is seldom reached by workers in a single discipline but requires coordinated efforts between many sciences.

In the development of research in recent years, two approaches to epidemiology have been pursued more or less separately. One group of investigators has been working from the clinical and experimental side, while another group has studied the actual distribution of diseases in the community in relation to the various factors and conditions which affect their distribution. Such differentiation in approach was necessary and has proven useful because the facts and inferences drawn from the two approaches to epidemiology extend knowledge more widely and establish principles relating to the phenomena of disease more solidly than if either approach had been followed exclusively.

The epidemiologist's contribution to the goal of prevention of diseases may be to piece together the known facts supplied by many disciplines into an hypothesis of the etiology of the disease. Or, he may, through field investigation, add to the known facts and develop an hypothesis of the etiology of the disease. Other investigators, working in other disciplines, may follow clues furnished by the epidemiologist and further test their validity. A classical example of a contribution to the prevention of disease that was made by following the epidemiological approach is that of the disclosure of the ability of fluorides to prevent tooth decay.[5] An epidemiological study of mottled dental enamel, after a long series of field studies, resulted in the disclosure of the inverse relationship between the amount of fluoride in a community water supply and the prevalence of dental caries. And now this information is used to reduce the incidence of dental caries in millions of children.

The definition, scope, functions, and goals of the science of epidemiology concern the study of periodontal disease as follows:

1. Epidemiological methods are applicable not only to the study of acute outbreaks of disease but also to investigation of chronic diseases such as diseases of the periodontal tissues.

2. Descriptive epidemiology describes the facts and conditions relating to the occurrence of a disease in groups of people. These facts and conditions which relate to the natural history of periodontal disease must be described if the links in the chain of events and conditions responsible for its initiation and progression are to be laid bare.

3. Constructive epidemiology goes beyond direct observations to development of hypotheses of cause and effect relationships. Such constructive efforts that are scientifically ordered will guide future investigations of periodontal disease.

4. The epidemiologist's goal is to furnish circumstantial evidence of all factors and conditions which may influence the disease process so that effective programs for prevention may be developed. Though many agents have been implicated as causes of periodontal disease, little headway has been made towards preventing the damage which they allegedly bring about. Every case of periodontal disease represents a failure at prevention.

The need exists for using epidemiological principles in gaining a better understanding of the natural history of periodontal disease. However, field studies of periodontal disease have been hampered by the lack of suitable methods for recording its oc-

currence and classifying its degrees of severity. Lord Kelvin has been quoted as saying, "Until you can count it, weigh it, or express it in quantitative fashion, you have scarcely begun to think about the problem in a scientific fashion."

The would be investigator of the epidemiology of periodontal disease is faced with the dilemma of either selecting one of the yardsticks already being tried or developing a new one. There are several basic principles which should be applied when developing or selecting a method of counting and quantitatively expressing periodontal disease.

NO SINGLE METHOD

A very significant point to consider is that probably no single method of classifying periodontal disease will ever be developed that will be appropriate for all types of research projects. The appropriate method is dependent upon the project objectives. These objectives must be clearly in mind before an appropriate method can be selected.

When the objective of a study makes it necessary to (1) measure small differences over a short period of time; (2) obtain a total assessment of the prevalence and severity of a condition, or (3) to record the disease in its incipient stage, then the diagnostic and recording procedures employed must be sufficiently detailed to produce these data. In such projects, it may be necessary to utilize all available diagnostic aids. However, as the techniques employed increase in complexity, more reliance must be placed on the examiner's skill and judgment, resulting in increased subjectivity. As subjectivity increases, so does the "between and within examiner" variability. The "between examiner" error may be of such magnitude in the more complex clinical studies that a single examiner is required for the entire study. Detailed, comprehensive studies, if correctly executed and the data properly analyzed, yield invaluable baseline information which cannot be obtained in any other fashion. However, the impracticability of using such complex methods when studying large segments of the population should be obvious. In many situations, it is more desirable to obtain less complete information on a more valid sample of a population group than to have such detailed information on a small segment which cannot be projected to represent the total population.

When the objective of a study is to disclose and describe differences in population groups, and when examinations from several investigators over a long period of time are to be compared, "between and within examiner" errors become of primary concern and must be reduced to a minimum. Objective criteria rather than subjective skill and judgment must be employed to ensure comparability of reporting. Objective criteria usually are developed by using simple procedures calling for a minimum of decisions which rely on the examiner's skill or judgment. This degree of simplicity and objectivity as a means of minimizing "between and within examiner" error is often obtained by limiting reporting to clear, positive, relatively advanced lesions. Underestimation of the total prevalence of the disease results, but acceptable examiner comparability is obtained. If the method utilizes objective criteria, the degree of underestimation should be sufficiently uniform to permit valid comparisons between and within groups. It should also permit calculation of the degree of departure from the baseline supplied by more comprehensive studies.

In diseases of low prevalence much may be learned by simply recording the presence or absence of a disease according to age, race, and sex. Other factors also may be associated with the disease, such as occupation, food habits, drinking water, climate, and social and economic environment. Such a separation of those who do and do not have evidence of alveolar bone loss would provide two groups whose characteristics could be studied carefully.

However, since periodontal disease is so common it is usually necessary to describe differences as differences in severity rather than prevalence. A method of classifying degrees of severity of periodontal disease to be suitable for epidemiological studies should have the following characteristics:

1. The method of choice must have definite, clear-cut criteria which reduce examiner decisions to a minimum. This characteristic is essential since epidemiological studies are primarily for the purpose of disclosing differences in prevalence and severity and for demonstrating the presence or absence of association between possible agents and the disease. Such objective criteria permit comparability of reporting by the same and other examiners.

2. The method should be sufficiently quantitative to provide a measure of the degrees of severity of the disease in a population group. The group index of severity is not an indication of each individual's need for treatment but is an indication of the average severity of the disease in the group or subgroups.

3. A method, to be practical for use in epidemiological studies of large populations, should have speed and simplicity of application rather than complexity even when the speed and simplicity require sacrificing some detail. This should not be construed as meaning to condone or promote "sloppy" or "slipshod" methods; rather it is an attempt to emphasize the need for using the simplest, most rapid method that will yield data pertinent to testing the hypothesis. An example is the dental caries index known as the DMF rate. The number of DMF teeth per person obtained by visual examination is an underestimation of the total since an additional number of carious teeth may be found by the use of x-rays.[6] Though it is an underestimation, the use of the DMF index together with an index of fluorosis enabled the fluoride-dental caries relationship to be disclosed. Although absolute data are not obtained by using the DMF rate, the level of measurement is high and relatively consistent, so that in many studies the added refinement offered by x-rays is not of practical value.

Another example of the use of simple methods is in low caries prevalence groups where just the per cent of persons having one or more DMF teeth is sufficient data from which to estimate the average number of DMF teeth per person for the group.[7] More recently it has been shown that the number of DMF tooth surfaces can be estimated accurately from the average number of DMF teeth per person.[8] These "time savers" permit the collection of data on a larger segment of the population in far less time than would be possible otherwise.

4. The techniques to be employed must be within the abilities of those employing them. Specialists in periodontia are rarely available for extensive field work; therefore, indexes for epidemiological studies of periodontal disease should be geared to the training and experience of the general practitioner.

5. The recordings should be suitable for reduction to a final score or index which is subject to statistical analysis.

Several approaches to the problem of evaluating systematically the condition of the periodontal structures have appeared in dental literature.[9-18] Each method has some desirable features as well as certain limitations. The actual or potential value of these methods cannot be predetermined but must be evaluated by retrospective analysis of their worth in specific epidemiological studies. However, when using any system one should keep in mind the type of data the particular method was designed to provide and the inherent limitations of the data so obtained.

An example of a system of classifying and scoring periodontal disease for field studies is that proposed by Russell.[13] In describing this method he says:

"The condition of the investing tissues is estimated individually for each tooth in the mouth, and is scored according to a progressive scale which gives relatively little weight to soft tissue inflammation and relatively great weight to destruction of bone. The score for an individual patient is the arithmetic average of the scores for the teeth in his mouth. The population score is the arithmetic average of the individual scores for the persons examined.

"Each tooth is scored according to the clinical condition of its supporting tissues. In the absence of overt inflammation in the free gingivae, or loss of function due to destruction of investing tissues, a tooth is considered to be negative and is assigned a score of zero. The scale provides for two stages of simple gingivitis, differing only in the extent of inflammation, and scored 1 and 2. (1—does not circumscribe the tooth, 2—completely circumscribes the tooth.) A score of 6 is assigned when a periodontal pocket is demonstrated, except when tissue destruction is so far advanced that masticatory function has been impaired; in this case the highest score, 8, is given."

This weighting system is designed to place greater emphasis on advanced disease and to minimize examiner disagreement. Throughout the scale the examiner is instructed to assign the lesser score when in doubt. This tends to cause underestimation but minimizes troublesome false positive recordings.

There has been only one clinical evaluation of this method reported in the literature. This evaluation was carried out by Russell on 200 patients at the National Institute of Health. These patients received both visual and x-ray examination.[13] On the basis of this clinical test, the investigators concluded that the field method of scoring is adequate for determining the relative status of the periodontal structures of two or more groups of populations, even though there is underestimation in each of the groups. The index described in this study appears to meet the minimum requirements for such an index. It is (1) quantitative, (2) simple, (3) rapidly applied, (4) reproducible, (5) subject to statistical procedures, and (6) can be used by the general practitioner after a short period of indoctrination. It must be kept in mind, however, that this method was designed for a specific purpose—that of comparing the severity of periodontal disease in various population groups. This method is particularly useful in studies (1) measuring large differences between groups, (2) of a single group on a longitudinal basis, or (3) when several examiners are participating in the study.

EXAMINER COMPARABILITY

In order to obtain examiner comparability, this method limits the reporting of periodontal disease to relatively late stages of gingivitis and pocket formation. The resulting underestimation of prevalence should be fairly uniform and according to Russell[13] the amount of underestimation can be computed by comparison with the more comprehensive clinical studies.

Some of the general characteristics of the epidemiology of periodontal disease which Russell's studies[19] have shown are:

1. The onset of periodontal disease may be at a very early age. The scores of prevalence and severity increase with age in more or less a straight line fashion if edentulous patients are excluded.

2. Periodontal disease is slightly more prevalent in males than in females. When present, it tends to be more severe in teen-age girls than in teen-age boys. The disease becomes more severe in males than in females after the mid-twenties or early thirties.

3. The Negro populations studied showed a higher prevalence and a greater severity of periodontal disease than whites at all ages.

By using this index of periodontal disease, Russell showed a greater severity of periodontal disease among 3,880 rural children in Indiana than he had previously reported for urban groups.[19,20] A review by Russell[21] of these and additional data indicates that the severity of periodontal disease is closely associated with an unrecorded factor. This factor becomes apparent when the data are grouped and analyzed according to the educational background of the community or of the individual. This factor is probably a reflection of a lack of adequate home and professional care. No difference was noted between the urban and rural residents when groups of similar educational background were compared. Russell and Ayers recently reported the findings from a study of 2,150 individuals in Birmingham, Alabama.[22] In this study, the Negro population had higher periodontal scores than the white persons of the same age, but when groups of equal education and occupational status were compared most of the difference in severity disappeared. Generally speaking, the fewer years of school completed by the adult the higher his periodontal scores tend to be. That this is probably in part a reflection of inadequate home and professional care is indicated by the direct association shown between the presence of obvious oral debris on two or more incisor teeth and higher periodontal scores.

In 1957 there was a limited study[23] of the epidemiology of periodontal disease in India in which Russell's method was used. These findings were compared with a similar study[23] in this country. These studies demonstrated a much greater severity of periodontal disease in the urban portion of the India study than was found in a group of urban children having the same age and sex in this country. Follow-up studies may help to explain these differences. Marshall-

Day reported a similar difference in severity between children in Northern India and Boston.[24]

Russell's method of classifying periodontal disease has been used in studies involving approximately 57 thousand individuals—some of these have been mentioned. There are other studies of the epidemiology of periodontal disease which are currently in progress or have been recently completed by the National Institute of Dental Research; for example, a longitudinal study of periodontal disease has been in progress since 1952 in the public schools in the State of Maryland.[25] It is hoped that by periodic examinations of the same subjects there will be documentation of what happens to the areas of gingivitis—whether they are transitory, persist as is, or persist and progress until there is destruction of the underlying tissues. Recently a study in nutritional and cultural patterns in relation to oral disease was conducted in Alaska. A similar study is now underway in Ethiopia. These investigations are coordinated with the Interdepartmental Committee on Nutrition for National Defense. The data collected from the Ethiopia study are expected to complement those from Alaska where ethnic, geographic, and climatic conditions, as well as dietary habits, appear to be in marked contrast with those seen in Ethiopia. A study of familial patterns of periodontal disease among Seventh Day Adventists has been underway for two years.

These epidemiological studies are designed to obtain information relating to the various factors and conditions which determine the occurrence and progression of periodontal disease. As stated previously, all of the elements necessary for the initiation and perpetuation of periodontal disease are in the agents, host, and environment. It is hoped that through proper design and execution of epidemiological and clinical investigations with careful attention to sample selection, controls, methods of collecting and analyzing data, that the phenomena surrounding these conditions and circumstances responsible for the initiation and progression of periodontal disease will be described sufficiently to make possible its effective prevention.

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SOUTHERN ACADEMY OF PERIODONTOLOGY ELECTS NEW OFFICERS

Left to Right: L. G. Roeling, member Executive Council; J. R. Owings, Secretary and Treasurer; L. F. Bumgardner, President, and F. T. Scott, President-Elect.

The Southern Academy of Periodontology recently elected the following officers for 1959: L. G. Roeling, member Executive Council; J. R. Owings, Secretary and Treasurer; L. F. Bumgardner, President; F. T. Scott, President-Elect.

The Annual Meeting of the Southern Academy of Periodontology will be held June 11, 12, and 13, 1959, at the Riverside Hotel, Gatlinburg, Tennessee. Guests are invited.

A Suggested Classification of Common Periodontal Disease

by FERMIN A. CARRANZA,* D.D.S. AND FERMIN A. CARRANZA, JR., D.D.S.,
Buenos Aires, Argentina

THE problem of a rational classification of periodontal disease based upon etiologic factors, pathologic changes and their clinico-radiographic manifestations has long challenged periodontists. This paper endeavors to clarify and facilitate the understanding of the intricate inter-relationships between the different factors which play a role in the etiology of periodontal disease and the microscopic and clinical response to them.

The term "periodontal disease" refers to all diseases which affect one or more tissues of the periodontium.[31] Under this heading *all types* of diseases affecting the gingiva and/or the supporting structures of the tooth are included.

The disease formerly called "alveolar pyorrhea" or "periodontoclasia" or "paradentosis" is by far the most common among periodontal diseases. According to the nomenclature accepted by the American Academy of Periodontology[31] it comprises the following conditions: periodontitis simplex, periodontosis, periodontitis complex, periodontal traumatism. According to the A.R.P.A. nomenclature it includes[1]: parodontopathia inflammata profunda, parodontopathia dystrophica, parodontopathia mixta.

Neither of the two above mentioned classifications have a term to design collectively all these conditions and similar in scope to "alveolar pyorrhea", "periodontoclasia" or "paradentosis." The correction of this omission in future nomenclatures appears highly desirable.

Some authors have coined terms of their own to refer to these conditions: Glickman uses "chronic destructive periodontal disease," Held uses "parodontolysis." This paper will be concerned *only* with this group which we shall provisionally name "common periodontal disease." Not included in this term are the purely gingival diseases and the so-called atrophies.

Common periodontal disease includes several syndromes, essentially chronic in character, which have two basic clinical manifestations: bone loss and pocket formation.

Bone loss is a sign without which a diagnosis of common periodontal disease cannot be made. In initial stages it is only microscopic. It may start in the alveolar crest affecting at first only the cortical layer, and proceed later to the underlying tissues reducing progressively the height of the alveolar bone. Or it may appear anywhere in the lamina dura, in isolated areas or generalized to the whole socket, widening the periodontal space.

Pocket formation is an extremely common manifestation, which, however, may be absent in initial stages of some cases.

All the local factors capable of producing or favoring pocket formation and bone loss are potential causes of common periodontal disease: they are the *determining factors,* which are necessary to initiate the lesions. These local causes fall into two categories:[5,6] those which produce *inflammation* and those which produce *trauma.*

The way in which bone is lost depends on the determining causes that induce its loss. The extension of inflammation from the pocket induces bone loss that starts in the alveolar crest and proceeds either horizontally or from the outer surface of the crest. Trauma produces bone loss anywhere in the alveolar bone proper or in the alveolar crest with a tendency to vertical shearing.

The characteristics and rhythm with which lesions progress depends on multiple factors of a constitutional and systemic

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kind which represent the *predisposing factor.*

We shall analyze these factors separately.

INFLAMMATION

Inflammation is a local tissue reaction to injury.[22a] The inflammatory response to local irritation in the gingiva produces the clinical picture of *gingivitis.* Systemic factors can modify or condition the gingival response.

The microscopic picture of gingivitis is characterized by the presence of an inflammatory infiltrate; epithelial changes which include proliferation, ulceration and degeneration; and a variable degree of proliferation of connective tissue fibers surrounding the inflamed area in an attempt to isolate it and stop its progress to the underlying tissues.

When the causes of inflammation are not severe and the fibrous structures of the gingiva are dense and strong, the inflammation can be isolated and "true" pocket formation does not take place. The lesion remains discrete, not invading the supporting tissues. This lesion constitutes an *inflammatory gingival syndrome,* clinically diagnosed as chronic marginal gingivitis. Although this syndrome may be—and usually is—a precursor to common periodontal disease it is not included under this term since it does not produce bone loss. It will not be considered in the following discussion.

When the causes of inflammation are severe and/or the fibrous structures are weak, inflammation may reach the supporting tissues, inducing an accelerated apical proliferation of the epithelial attachment with the formation of a "true" or "absolute" pocket and loss of alveolar bone and periodontal membrane. The presence of inflammatory cells within the marrow spaces or close to bone surfaces disturbs the delicate balance which exists between bone formation and bone destruction, resulting in bone loss. All these features characterize the *inflammatory periodontal syndrome.*

When the reparative capacity of bone is normal or favorable, bone loss is only superficial constituting what may be called *superficial inflammatory syndrome.* The inflammatory infiltrate does not burrow deep, bone loss is slight and active areas of repair are seen microscopically. Its progress is very slow.

In other cases, the defensive capacity against irritation and/or the reparative capacity of bone are poor or unfavorable. Bone loss is then severe and the inflammation penetrates rapidly. This picture may be called *deep inflammatory syndrome.*

The clinical picture produced by the inflammatory periodontal syndrome is characterized by supraalveolar suprabony pocket formation, gingival bleeding, suppuration, subgingival calculus, horizontal bone loss. Tooth mobility is a late sign. Clinically the superficial inflammatory syndrome has shallower pockets and a slower progress than the deep inflammatory syndrome.

Local irritational factors capable of inducing an inflammatory reaction are well known. They include materia alba, calculus, food impaction, overhanging fillings, etc. The normal tissue reaction to these factors is conditioned by systemic factors that control the reaction of the periodontal structures. Unfavorable systemic factors may facilitate the extension of inflammation to the supporting tissues and increase the rate of bone loss locally initiated. Glickman[8,12] and Stahl et al.[33] have shown in experimental animals that lesions produced by local irritants are more severe in the presence of systemic disturbances.

Conditions which may reduce tissue resistance to inflammation include hormonal, nutritional, gastro-intestinal, infectious disturbances.[11,15,22,24]

TRAUMA

Traumatizing factors produce the *traumatic periodontal syndrome.*

Trauma favors bone loss and disorganization of the periodontal membrane and interferes with the normal reparative function of these tissues. Although it does not seem to initiate pocket formation,[3,13] it can contribute to pocket deepening by weakening the fibrous gingival system, thus facilitating the penetration of inflammation.

In the first stages of trauma, lesions are produced mainly by compression which leads to hemorrhages and thrombosis of the blood vessels and necrosis of areas of periodontal membrane.[3,13,18,25] But if the traumatic influences are not extremely severe and the periodontal structures are reasonably strong, these lesions can be repaired. They are *compensated traumatic lesions* which have no clinical manifestation except a slightly increased tooth mobility at bite pressure and sometimes thickening of the lamina dura and/or apical periodontal space. They are very seldom diagnosed.

But traumatic forces can exceed the reparative potentialities of the supporting tissues due to the former being extremely harmful (due to unfavorable direction, excessive intensity or excessive frequency) or the latter being reduced due to systemic factors. Stahl et al.[34] have shown in experimental animals that vertical occlusal trauma causes degenerative changes in the periodontal membrane, which are reversible in animals fed a normal diet, and not repaired in animals subjected also to protein deprivation.

When the reparative capacity of bone is insufficient to compensate the destruction produced by traumatic forces, *uncompensated traumatic lesions* begin to appear.

These uncompensated traumatic lesions are manifested in sides of tension and pressure. They are areas of resorption which are insufficiently repaired or not repaired, leading to osteoporosis of the cortical layer; this in turn produces widening of the periodontal space and disorganization of the periodontal membrane.[2,7,25,27,37]

Clinically these lesions are manifested by tooth mobility, and eventually by migration of teeth and formation of diastemas without pocket formation. Radiographically by widening of the periodontal space and fuzziness or absence of the lamina dura, reduction in height of the alveolar crest with tendency to vertical shearing.

Traumatic forces are produced mostly by occlusal disharmonies but also by pressures of soft tissues, tongue thrusting, etc. Their frequency and intensity can be increased by oral habits such as bruxism, lip biting, etc. These habits may be produced or favored by systemic factors such as toxic, nervous or psychosomatic disorders. This group of systemic factors plays an indirect role in the etiology of traumatic lesions by favoring local factors.

COMBINED LESIONS

There is a third category which is a combination of the other two and shares in different degrees their characteristics, constituting the *combined periodontal syndrome.*

When both trauma and inflammation act upon the periodontal structures but their deleterious action remains below the threshold of resistance of the tissues, the clinical result will be a chronic marginal gingivitis or an inflammatory superficial syndrome plus a slightly increased tooth mobility due to the presence of compensated traumatic lesions. This combination may be called *compensated combined periodontal syndrome.*

When trauma and inflammation act upon weakened periodontal structures going beyond the threshold of resistance, the result will be a case of periodontal disease with both uncompensated traumatic lesions and inflammatory lesions, an outstanding feature of which will often be the presence of intraalveolar pockets. These lesions characterize the *uncompensated combined periodontal syndrome.*

Clinically this syndrome is recognized by tooth mobility, deep pocket formation, pockets of uneven depth, frequently of

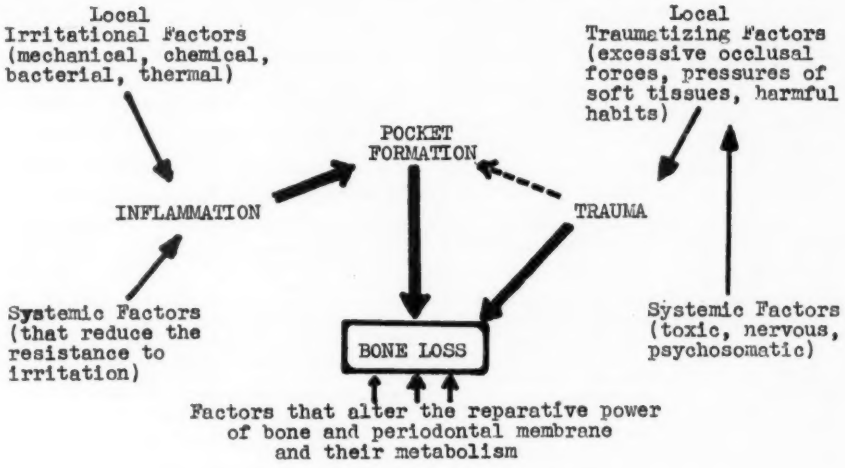

Figure 1 - Etiologic Chart (Carranza[5,6])

the intraalveolar (infrabony) type, calculus, suppuration, eventually migrations and diastemas. The latter are the result of traumatic forces acting against the teeth constantly in one direction, as is the case with tongue thrusting.

Radiographically the uncompensated combined syndrome reveals widening of the periodontal space, localized or generalized loss of lamina dura and vertical bone loss.

PREDISPOSING CAUSES

Under this heading all the causes which alter the reparative capacity of bone and periodontal membrane and their metabolism are included. They are identified with Glickman's "bone factor" which has been defined as the "systemic regulatory influence upon the response of alveolar bone to local factors."[9,10] Valuable research papers by Glickman and his co-workers have given a solid background to the bone factor concept, which constitutes the predisposing factor of common periodontal disease.

The interrelation between local and systemic factors has been very clearly shown by Gratzinger[19] in a graph which demonstrates how tissue resistance and local factors can vary in the different cases of periodontal disease.

The normal metabolism of the supporting tissues can be impaired by different systemic factors which ultimately may produce either or both of the following: (a) a deficiency in the mechanism of bone formation, or (b) an exaggeration in the mechanism of bone resorption, favoring in this way the loss of bone.

According to Schnitman[32] systemic causes of bone loss can be classified as follows:

I. The mechanism of bone formation may be altered by:

A. A defect in osteoblastic activity due to lack of estrogens, lack of thyroid hormone, lack of androgens, lack of somatotrophin, lack of function.

B. A defect of the bone matrix due to lack of proteins (primary or secondary) or to a defect in collagen formation due

Etiology		Pathology (Microscopic picture)	Clinico-Radiographic Picture	Name Suggested	
Determining factor	*Reparative tissue reaction*				
INFLAMMATION	Normal	Similar to the following but with slight or no deepening tendency. Normal reparative bone processes.	Gingival inflammatory changes. Relative pockets. Shallow true pockets. Slight horizontal bone loss. Very slow evolution.	INFLAMMATORY PERIODONTAL SYNDROME	SUPERFICIAL
	Poor	Inflammatory infiltrate. Epithelial proliferation. Accelerated apical migration of the epithelial attachment. Extension of inflammation to supporting tissues. Denudation, necrosis and breakdown of cemental pocket wall. Calculus. Osteoclastic resorption of alveolar crest. Marked tendency to horizontal bone loss. Suprabony pocket formation.	Gingival inflammatory changes. Bleeding, calculus, suppuration. Supraalveolar pockets. Horizontal bone loss with loss of lamina dura at the alveolar crest. Mobility: Late or absent.		DEEP
TRAUMA	Normal	Thrombosis, necrosis of areas of periodontal membrane. Localized resorption of bone and cementum. Compensatory repair of resorbed areas.	Slight mobility to occlusal pressure. Sometimes thickening of the lamina dura and apical periodontal space. No pocket formation.	TRAUMATIC PERIODONTAL SYNDROME	COMPENSATED
	Poor	Osteoporosis of the lamina dura. Widening of the periodontal space. Disorganization of periodontal fibers. Reduction in height of alveolar bone with marked tendency to vertical bone loss. Slight or no compensatory repair of resorbed areas.	Mobility. Eventually migrations and diastemas. Widening of the periodontal space. Localized or generalized loss of lamina dura. Vertical bone loss. No pocket formation.		UNCOMPENSATED
COMBINATION	Normal	Compensated traumatic lesions + superficial inflammatory lesions.	Inflammatory gingival picture + compensated traumatic picture. Shallow pockets.	COMBINED PERIODONTAL SYNDROME	COMPENSATED
	Poor	Uncompensated traumatic lesions + deep inflammatory lesions, frequently intraalveolar pockets.	Uncompensated traumatic picture + deep and uneven pockets, frequently intraalveolar. Marked mobility.		UNCOMPENSATED

to vitamin C deficiency (primary or secondary).

C. A defect in mineralization and enzymatic factors due to primary calcium deficiency, inadequate Ca/P ratio, lack of magnesium or vitamins, increased calcium or vitamin D excretion.

II. An exaggeration in the mechanisms of bone resorption as a result of hyperparathyroidism, acidosis, renal failure, osteolytic neoplasms.

These systemic causes constitute the real internal predisposing factor of common periodontal disease. They are rarely clinically evident and it is probable that in most cases they remain below levels demonstrable by present-day diagnostic techniques. It is the addition of local factors that brings periodontal changes within clinically detectable levels.

RECAPITULATION

According to the above discussion, etiologic factors of common periodontal disease are classified as follows:

A. Determining factors: Inflammation and Trauma.

B. Predisposing factors: All the factors which alter the reparative power of bone and periodontal membrane.

Determining factors are local in origin but their action can be aggravated by systemic factors which act either by (a) interfering with the mechanism of inflammation, modifying the usual gingival response to local irritation; these are the systemic conditioning factors such as diabetes, vitamin deficiencies, etc. (b) by increasing the intensity or frequency of local traumatizing factors. These are the toxic, nervous and psychosomatic disorders.

Predisposing factors are of systemic origin. They act by altering the normal metabolic interchanges of bone and periodontal membrane and the reparative capacity of the tissues.

It is important to note that a given condition can act in more than one way. For example, vitamin C deficiency acts as a conditioning factor by reducing the strength of the fibrous gingival attachment thus interfering with the defensive tissue reaction and favoring the propagation of inflammation to the underlying tissues. At the same time it is a predisposing factor that affects the mechanism of bone formation by interfering with bone matrix deposition. Glickman's experiment on the interaction of vitamin C deficiency and artificially induced inflammation showed that "acute vitamin C deficiency alters the response of the periodontal tissues so that the destructive effect of inflammation is accentuated. The exaggerated destruction in the presence of inflammation results partly from an inability to marshal a defensive delimiting reaction to the inflammation and partly from destructive tendencies caused by the deficiency even in the absence of inflammation."[8]

Predisposing factors are seldom diagnosed, but they favor or increase the destruction produced by local factors. Predisposing factors do not by themselves unbalance the equilibrium between bone formation and bone destruction (except in very unusual cases) if local factors—inflammation and trauma—are not added.

Comment on periodontosis: The existence of the pathological and clinical entity "periodontosis" has long been discussed in periodontal literature. One group of authors[14, 16, 17, 26, 36] describe periodontosis as a primary degenerative disease of the periodontium which in a late stage can become complicated with the inflammatory changes of pocket formation.

Other investigators[20, 21, 23, 28, 29] believe that the histopathologic changes described as characteristic of periodontosis are produced by trauma. The viewpoint expressed in this paper adheres to the latter school of thought. The pathologic changes of periodontosis, described as dystrophic or degenerative can be produced by a com-

bination of traumatizing forces acting upon weakened periodontal structures. Ramfjord[28] has shown that the healing stage of traumatic lesions may show features which resemble the commonly accepted picture of periodontosis. Stahl et al.[34] have shown degenerative changes in the periodontal membrane of experimental animals due to vertical occlusal trauma.

According to the classification suggested in this paper, the first two stages of periodontosis would be classified as "uncompensated traumatic syndrome," which would become "uncompensated combined syndrome" when pocket formation ensues.

Early mobility, migration and diastemas are the result of local traumatizing factors superimposed upon tissues weakened by systemic factors. Of great importance among traumatizing factors in these cases are tongue thrusting,[30] cheek and lip biting pressures, bruxism and other habits, impediments of lateral gliding[4,35] and others, sometimes not easily discovered.

SUMMARY

An attempt has been made to explain the role of the different local and systemic factors in the etiology of common periodontal disease. The interrelation of local and systemic factors is explained and a classification of common periodontal disease is presented.

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THE AMERICAN INSTITUTE OF DENTAL MEDICINE

The 16th Annual Meeting of The American Institute of Dental Medicine will be held in Palm Springs, California, November 8-12, 1959.

Dr. Franz Alexander, Director of the Psychiatric and Psychosomatic Research Institute of Mount Sinai Hospital in Los Angeles, will discuss responses to fear and other emotional stress situations as they occur in life, with special reference to emotional factors in chronic diseases.

Dr. Hermann Becks, the President of the Institute, will report on dentistry and dental research as it is in Europe today

Dr. Douglas Gordon Campbell will lecture on the personal factors involved in malpractice suits; the psychopathology of nutrition; and personal hazards of dentistry.

Dr. Thomas K. Cureton, Professor of Physical Education and Director of the Physical Fitness Research Laboratory at the University of Illinois will give good information to the practitioner regarding basic principles of physical fitness and methods of improving and maintaining good health.

Dr. Marie U. Nylen, from the National Institute of Dental Research, Bethesda, Maryland, will describe her work with electron microscopic studies with dentinogenesis, amelogenesis, and calcification.

Dr. Max Sadove, Professor of Surgery (Anesthesiology) at the Research and Educational Hospitals of the University of Illinois, will "continue where he left off" two years ago in the field of anesthesiology and the applied aspects of pharmacology.

Dr. Reidar F. Sognnaes, Assistant Dean of the Harvard School of Dental Medicine, will present material concerning dental erosion, cornification and desquamation of oral mucosa, and mineralization, demineralization, and remineralization.

Dr. Stafford L. Warren, Dean of the School of Medicine of the University of California at Los Angeles, will discuss the effects of radioactive fallout on the human body and describe the formation of fallout, treatment, precautions, and therapeutic doses.

Lysozyme Content of Human Gingiva and Various Rat Tissues

by GEORGE W. BURNETT, SUSAN GOUGÉ, AND A. E. TOYE, *Washington, D. C.*

LYSOZYME occurs in several secretions of the human body such as tears, nasal mucus, gastric juice and saliva where, due to its mucolytic activity, it may greatly influence the composition of the so-called saprophytic species of the microbial flora of the contiguous areas, particularly the oral microbial flora (see reviews of Thompson[1] and Simmons[2]). Since lysozyme undoubtedly has a significant role in the defense mechanism of the body, it could indirectly influence those forms of periodontal disease which are mediated in part by the oral microbial flora.

Lysozyme also occurs in tissues in varying quantities and several theories have been advanced to account for its presence and activity. Investigations have been made of the possible relationship between lysozyme and vitamin A but it was found, contrary to the initial assumption, that lysozyme and vitamin A are independent factors in the defenses of the rat against infection. In avitaminosis A excessive lysozyme may be produced to compensate for vitamin deficiency.[3] More lysozyme has been found in the tissues of the first part of the duodenum and less was found in the fundus, particularly in individuals with ulcers, signifying a possible etiologic relationship between lysozyme activity and peptic ulcers.[4,5] A relatively large amount of lysozyme has been found in the mucosa of the colon and in the stools of individuals with ulcerative colitis. The lysozyme content of the mucosa is greatest in epithelial tissue, indicating a possible etiological relationship to ulcerative colitis.[6,7] In contrast to epithelial tissues, the lysozyme content of most mesodermal tissue is low but granulation tissue contains considerable lysozyme (up to 500 units per gram).[8] It is evident, therefore, that the amount of lysozyme increases in tissues in response to irritation and inflammation and under certain circumstances may be related to the development of ulcerative lesions. Periodontal tissues respond to bacteria and other irritants, oftentimes prolonged and persistent, by the process of inflammation. Since the substrate of lysozyme is present in periodontal tissues, it was of interest to determine the lysozyme content of inflamed gingival tissues, removed during gingivectomies, for comparison with the lysozyme content of other tissues.

From the Division of Dentistry, Walter Reed Army Institute of Research.

EXPERIMENTAL METHODS

Normal gingival tissue, kidney, spleen, and liver from each of 9 white rats were assayed for lysozyme content according to the method of Lobstein and Fogelson.[9] Fourteen specimens of inflamed human gingival tissue, removed during gingivectomy, were also tested for lysozyme content.

The animal tissues were removed immediately after the death of the animals, which were killed by an overdose of nembutal. The human gingival tissues were collected in a vessel refrigerated with dry ice immediately after surgical removal. All tissues were kept frozen until they were tested, which was usually immediately.

The tissue samples were weighed, then homogenized with 10 ml. of cold phosphate buffer, pH 6.2, in a cold Waring blendor. The homogenate was centrifuged at 2000 rpm for one hour in a refrigerated centrifuge at 0^0-1^0C. The supernatant fluid was pipetted into a sterile 100 ml. volumetric flask. A second 10 ml. of buffer was added to the tissue sediment which was again homogenized in the Waring blendor and centrifuged for 30 minutes. The supernatant from the second extraction was added to the supernatant from the first extraction. The process was repeated for a third time and the supernatant

TABLE I. Lysozyme Content of Certain Rat Tissues

Test Number	*Gingiva*		*Kidney*		*Spleen*		*Liver*	
	%	*ug/g tissue*	%	*ug/g tissue*	%	*ug/g tissue*	%	*ug/g tissue*
1	0.26	2,603	0.042	421	0.011	112	0.0	0.0
2	0.67	6,750	0.03	307	0.013	132	0.0026	26.5
3	2.44	24,453	0.064	642	0.0047	47	0.0	0.0
4	3.18	31,810	0.062	615	0.002	21	0.008	81.6
5	0.21	2,146	0.0196	196	0.013	133	0.008	88.9
6	2.78	27,857	0.0277	277	0.035	354	0.002	21.7
7	1.89	18,918	0.026	266	0.014	148	0.006	61.4
8	1.92	19,197	0.025	253	0.0249	249	0.004	42.1
9	2.50	25,222	0.035	351	0.027	272	0.0037	37.7
Mean	1.76	17,662	0.0368	370	0.0161	163	0.0038	40.0
Standard Error of Mean	0.336	3,769	0.0047	47.64	0.0033	35.56	0.00090	9.62
Coefficient of Variation	66.6	74.9	40.5	40.6	61.2	65.6	71.8	74.7

fluid pooled with the previous supernatants. The volume in the flask containing the three supernatants was then brought up to 100 ml. with the buffer and the test was made with the diluted extracts.

In determining the lysozyme content of tissue by the method of Lobstein and Fogelson,[9] lysozyme utilized for the standard was crystalline egg white lysozyme obtained from Difco. The standard suspension of *Micrococcus lysodeikticus* was prepared from ultraviolet-killed and desiccated bacterial cells obtained from Difco.

RESULTS

The results of determining the lysozyme content of the gingiva, kidney, spleen and liver of each of 9 rats are shown in Table 1 as the percentage of the tissue and as ug per gram of tissue. The gingiva of the rat contained considerably more lysozyme than did any of the other tissues tested, with an average of 17,662 ug per gram of tissue in comparison to an average of 370, 163 and 40 ug per gram of tissue respectively for kidney, spleen and liver. However, there was considerable variation in the determined lysozyme content of the various tissues. In the case of the rat gingiva, the range of lysozyme extended from 2,146 ug per gram of tissue to 31,810 ug per gram of tissue, with a coefficient of variation about the mean of 75 per cent. While the range of values for gingiva and other tissues was considerable, there was some indication of a high and low range, although many more observations would have to be made in order to demonstrate such a trend. There can be little doubt, however, that there is more lysozyme in rat gingiva than in rat kidney, spleen or liver. The mean percentage lysozyme content of rat gingiva, kidney, spleen and liver was respectively 1.76 per cent, 0.036 per cent, 0.016 per cent and 0.0038 per cent.

The lysozyme content of human gingival tissue from 14 gingivectomies is shown in Table 2 for comparison with the lysozyme content of rat tissues. Human gingiva contained an average of 1,493 ug per gram of tissue in comparison to approximately 17,000 ug per gram of rat gingiva. The range of lysozyme content of human gingiva was considerable, even more so than with rat gingiva, extending

TABLE II. Lysozyme Content of Human Gingival Tissue

Test Number	*ug per gram tissue*	*%*
1	44	0.004
2	269	0.026
3	99	0.009
4	7,276	0.727
5	9,036	0.903
6	279	0.027
7	333	0.033
8	278	0.027
9	522	0.052
10	2,219	0.220
11	137	0.014
12	310	0.031
13	43	0.004
14	62	0.006
Mean	1,493	0.15
Standard Error of Mean	772.04	0.077
Coefficient of Variation	193.8	192.7

from 43 ug per gram of tissue to 9.036 ug per gram of tissue with a coefficient of variation of 193 per cent. The mean percentage composition of human gingival tissue was 0.15 per cent in contrast to 1.6 per cent for rat gingival tissue.

DISCUSSION

As indicated previously[1-7] lysozyme may increase in tissue in response to irritation and inflammation and be in some way related to the breakdown of tissues and development of ulcers. Since there may be an increase in lysozyme in inflamed tissue, it would have been suspected that inflamed human gingival tissues would contain more lysozyme than would "normal" rat gingival tissue, but there was, on the average, slightly more than 11 times more lysozyme in rat gingiva than in human gingiva. It is difficult, however, to assess the significance of the lysozyme concentration in human gingival tissue because no normal lysozyme values have been established. Because of the difficulty of obtaining "normal" gingival tissue, it will be difficult to establish the lysozyme content of such tissue. The possible relationship of lysozyme to periodontal disease is also confused by the findings of Joseph and Shay[10] of a decrease in salivary lysozyme of individuals with periodontal disease. A simultaneous determination of both the lysozyme in saliva and gingival tissue might be helpful.

In Table 3, a comparison is made between the present data and that of other investigators with regard to the lysozyme concentration of rat tissues. Since in each investigation different methods of determination and reporting were used, a comparison of the values is difficult if not impossible. Only a proportionate trend can be made in the lysozyme content of different tissues as reported by different investigators.

SUMMARY

An investigation has been made into the lysozyme content of normal rat gingiva, spleen, kidney and liver and inflamed hu-

TABLE III. Comparison of Lysozyme in Rat Tissues According to Various Investigators

Reference	*Units*	*Kidney*	*Spleen*	*Liver*	*Respective Proportional Distribution*
Smolelis and Hartsell[11]	ug/ml extract	24.0			
Cardinali[12]	Fleming units/g tissue	8,800	8,530	470	22:21:1
Litwack[13]	ug/mg N tissue	87	37.2	5.3	17:7:1
Burnett and Gougé	ug/g tissue	360.1	171.2	40.7	9:4:1

man gingiva obtained from gingivectomies. Normal rat gingiva (17,662 ug/g.) was found to contain an average of 11 times more lysozyme than inflamed gingiva (1,493 ug/g.). On an average, rat kidney (370 ug/g.) contained more lysozyme than did rat spleen (163 ug/g.) which contained more lysozyme than did rat liver (40 ug/g.).

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Book Review . . .

Dental Biographical Sketches. By A. Porter S. Sweet, D.D.S., F.A.A.O.R. The Naportan Co., Pittsford, New York. 1958. Price $2.00.

Dr. A. Porter S. Sweet, Editor of Dental Radiography and Photography, has since 1946 written and published dental biographical sketches on the inside front cover of the magazine he edits. These biographical sketches have now been collected into a neat paper backed brochure of 32 pages and cover.

The biographies are of men who have been prominent in the field of dentistry and include Josiah Flagg, Solyman Brown, G. V. Black, Wallace Clyde Davis, Willoughby D. Miller, James Leon Williams and Pierre Fauchard. Also, a short summary of the career of Saint Apollonia is included.

The material about each of these individuals is interestingly presented and apparently authoritative. All interested in dental history will want to have a copy of this pamphlet.

Presence of Acetylcholinesterase in Human Gingiva

by JAMES K. AVERY AND ROBERT RAPP, *Ann Arbor, Michigan*

NEURAL receptors of pressure, pain, touch and temperature have long been known to exist in the tissues surrounding the teeth. Recently, by means of the Power's modification of Romane's method[1] of impregnation with silver nitrate, Rapp, Kirstine and Avery[2] confirmed earlier work[3,4,5,6] regarding the presence of neural terminations in the human gingiva.

Acetylcholine (ACh) is now believed to play an essential role in the transmission of impulses along neural fibers and across synapses.[7] According to Nachmansohn,[8] ACh functions in alteration of membrane potential by means of increasing ion permeability of the nerve fiber membrane which then functions in the process of generating a bioelectric potential. Associated with neural impulse transmission is hydrolysis of the ACh by specific or acetylcholinesterase (AChE) which terminates neural activity. In 1949 Koelle and Friedenwald[9] developed a histochemical method for demonstration of cholinesterase (ChE) activity. The presence of ChE is believed to be indicative of the presence of ACh. Subsequently, Koelle,[10,11] Pearse,[12] Churchill, Schuknecht and Doran[13] and Avery and Rapp[14] have presented modifications and improvements on this technique. It is the purpose of the present study to apply this technique to oral tissues to determine the presence and location of a specific ChE system in the human gingiva.

METHODS

The technique of Koelle and Friedenwald[9] consists of incubating tissues containing the enzyme ChE in the presence of acetylthiocholine (AThCh) and copper ions. The resulting precipitate of copper thiocholine (CuThCh) is subsequently treated with ammonium sulfide to produce a brownish copper sulfide precipitate at the sites of enzymatic activity.

Fifty samples of fresh gingival biopsy tissue were obtained and divided into two series. The specimens of the first series were immediately frozen and sectioned on a freezing microtome and mounted on albuminized slides. The gingival tissues of the other series were trimmed into blocks of approximately 3 cubic mm. and placed in the storage solution. Each series of gingival tissues was then divided into four group of tissues, was incubated without a ner. The *first group* of tissues was treated with 10^{-6}M di-isopropylfluorophosphate (DFP)* for thirty minutes at 37°C to inactivate the nonspecific ChE and then incubated with AThCh for 17 hours to reveal the sites of activity of only the specific ChE. The *second group* of tissues was incubated in a substrate of butyrylthiocholine (BuThCh) for 17 hours at 37°C to indicate the sites of only nonspecific ChE. The *third group*, a control group of tissues, was incubated without a substrate, and tissues of the *fourth group*, also a control, were treated with DFP and then incubated with BuThCh. The tissues of all groups were then treated with ammonium sulfide and the resulting brown precipitate of copper sulfide revealed the sites of ChE activity. The sectioned tissues of the first series were fixed in formalin, dehydrated, counterstained with eosin and mounted. The blocks of gingival tissues were fixed in formalin, dehydrated, embedded in paraffin, sectioned at 10 to 30 micra, counterstained with eosin and mounted.

This investigation was supported by the Research and Development Division, Office of the Surgeon General, Department of the Army under contract No. DA-49-007-MD-766 at the University of Michigan School of Dentistry.

*Di-isopropylfluorophosphate was generously supplied by Dr. William H. Summerson of the U. S. Army Chemical Warfare Laboratories, Army Chemical Center, Maryland.

Fig. 1. Specific acetylcholinesterase in human attached gingiva. Counterstained with eosin. The AChE appears to be localized along nerve fibers in the connective tissue papillae underlying the gingival epithelium.

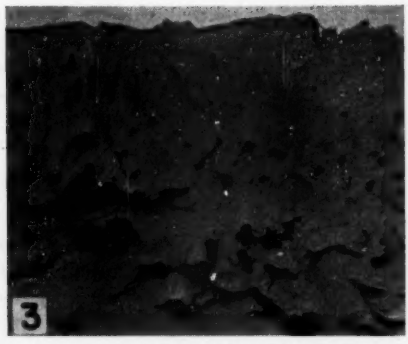

Fig. 3. Non-specific cholinesterase appearing in the blood vessels of the lamina propria of human gingiva.

FINDINGS

Upon examination of tissues of group one, specific ChE was found present in the connective tissue papillae underlying the gingival epithelium. The specific ChE appeared concentrated along fine fibers which could be traced from deep in the reticular layer of the lamina propria and into the connective tissue papillae, Fig. 1. Some of the AChE-positive fibers appeared to form loops and coils within the papillae, Fig. 2.

In the tissues of group two which were incubated in BuThCh substrate, non-specific ChE was found in the blood vessels throughout the lamina propria, Fig. 3. The specific and non-specific ChE appear, thus, in similar locations in the gingiva. This is to be expected since the nerve fibers are closely associated with the blood vessels. It can be observed that the non-specific ChE delineates the larger diameter blood vessels whereas the specific ChE is seen in the fine, smaller diameter nerve fibers.

Examination of tissues in the third group incubated without the thiocholine substrate revealed no staining in the gingival tissue, Fig. 4. Tissues in group four treated to eliminate the non-specific enzyme and then

Fig. 2. Specific cholinesterase in coiled fiber of connective tissue papilla. Oral epithelium overlies the connective tissue papilla.

Fig. 4. Control tissue incubated without substrate. Absence of staining in gingival tissues.

incubated to reveal only this enzyme also showed no evidence of staining. No difference in results was obtained between the series of tissues sectioned before and the series sectioned after incubation and staining.

DISCUSSION

The technique of Koelle and Friedenwald[9] for the location of ChE is applicable to the study of human gingiva. Biopsy tissues incubated immediately after sectioning on the freezing microtome as well as blocks of tissues sectioned after incubation both reveal the presence of specific and non-specific ChE. It is believed that false localization of the enzymes was controlled because 1) all biopsy material was utilized immediately after procurement, 2) high concentrations of sodium sulfate were placed in the storage and incubation solutions and 3) the incubation solutions were saturated with copper thiocholine. The latter two techniques have been found successful in eliminating enzymatic diffusion which had previously resulted in artifacts of localization.[11] It is of interest that a specific ChE system is present in the connective tissue underlying the gingival epithelium in the same location where investigators such as Lewinsky and Stewart,[4] Bradlaw,[5] Gairns and Aitchison[6] and Rapp, Kirstine and Avery[2] have demonstrated specialized receptors of pain, touch and temperature.

A specific ChE system has been noted previously in sensory nerves by Koelle,[15] Hebb,[16] and Avery and Rapp.[14] An incubation time of 17 hours was found necessary to hydrolyze the available substrate in the gingiva and thus to demonstrate the presence and location of the enzyme. Koelle,[11] on the other hand, found incubation for 60 minutes sufficient to demonstrate ChE in axons, dendrites and cell bodies of cholinergic neurons. It is probable then that concentrations of the enzyme are considerably less in the sensory nerve fibers of the gingiva than in parasympathetic nerves.

CONCLUSIONS

1. Specific ChE is present in the connective tissue papillae underlying human gingival epithelium.

2. Non-specific ChE is present in concentrated amounts in blood vessels located throughout the papillary and reticular layers of the lamina propria of the human gingiva.

The authors wish to express their sincere appreciation to Miss Elizabeth Haley for her technical assistance.

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THE NORTHEASTERN SOCIETY OF PERIODONTISTS

The new officers of the Northeastern Society of Periodontists are:

President, Harold J. Leonard; President-elect, George J. Witkin; Secretary, Irwin W. Scopp; Treasurer, George J. Whinston; Editor, Norman E. Alderman; Chairman Committee on Membership, Charles L. Berman, and Chairman Committee on Education and Publicity, Anthony F. Posteraro.

Plans are under way for a fall seminar in Periodontology. For details, write Anthony F. Posteraro, D.D.S., Chairman, Committee on Education and Publicity, 43 Fifth Ave., New York 3, N. Y.

THE AMERICAN INSTITUTE OF DENTAL MEDICINE CHANGES NAME

The growing demand and the great emphasis in the teaching areas of the basic sciences led to the establishment in 1954 of a Division of Oral Biology at the University of California which includes the Sections of Oral Histology, Oral Pathology, Oral Medicine, and Oral Diagnosis. From the increasing demand for post graduate education in the borderline fields of dentistry and medicine and the health sciences, it appears that the name of the American Institute of Dental Medicine is not all inclusive. At a recent meeting of the Board of Directors the name was therefore changed to: *The American Institute of Oral Biology.*

The effective date for the change is July 1, 1959.

Bruxism Splint

Technique Using Quick Cure Acrylic

by HAROLD E. GRUPE, D.D.S.,* AND JOHN J. GROMEK,** *U. S. Army Europe*

THE need for protective splints in bruxism cases has long been recognized. However, the clinical problem of constructing such a splint that would satisfy the basic requirements including retention, durability, comfort, minimum adjustment and patients acceptance is not an easy one.

GENERAL CONSIDERATION OF PROBLEM

Many bruxism splints have proven to be orthodontic appliances; others have produced damage to the palatal gingiva. Frequently some teeth involved in the support or retention of the splint become sore or loose. Furthermore, if the splint is tissue-borne, it may settle; and does not maintain a predetermined relationship between the upper and lower teeth.

It would seem reasonable that such a problem demands a splint which is completely tooth-borne. The quick cure acrylic materials offer the possibility of an absolutely accurate fitting of such an appliance; with none of the problems previously encountered in curing or casting cases to a model or die, and later fitted to a master model or to the teeth themselves.

*USAREUR Consultant in Periodontia and Chief of the Periodontia Section at the Vogelweh Dental Clinic, APO 227, US Forces.

**Chief Dental Lab. 570 Med Det (Dent Pros Mble) APO 227, US Forces.

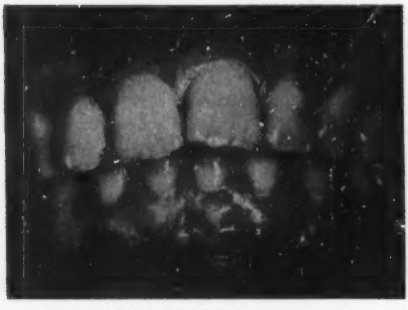

Fig. 1. Normal clinical appearance.

Fig. 2. Pathologic attrition for age 20.

Fig. 3. Occlude casts to determine width of overjet.

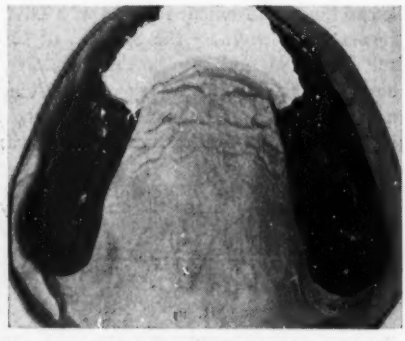

Fig. 4. Adapt a lower baseplate to level of highest contour of crowns. Add plaster core.

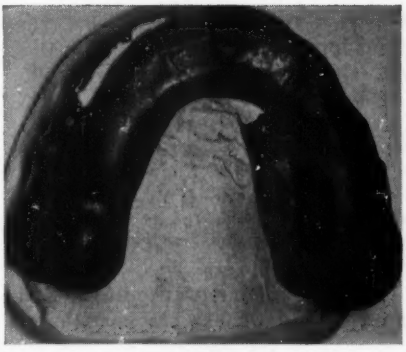

Fig. 5. Adapt second baseplate over the first one. This is the tray. Mark mid-line.

Fig. 7. Baseplate removed with hand torch and chipping away pieces. Splint inserted; checked with carbon and spot ground for maximum contact with lower anterior teeth only.

ADVANTAGES OF METHOD

The comfort and retention of this quick cure acrylic appliance can be consistently relied upon, without aid of clasps or wires. The time required to make such a splint is unusually short (2-3 hours); and the cost is small. The simplicity of the technique affords the possibility of wide usage. Steps in technique of construction are shown in pictures with legends.

PRACTICAL CASE PRESENTED

The case shown here is that of a twenty year old soldier who presented no dental complaints. However, he stated that several men who slept in the same barracks were disturbed almost nightly by the noise from his teeth. The excessive wear on the incisal edges is striking and would indicate the habit is of long standing. The history was negative for occupational contributing factors. The roentgenograms showed no evidence of pathology. The gingiva appears clinically normal.

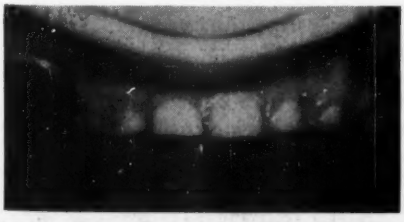

Fig. 6. Tray inserted, filled with quick-cure acrylic. Hold only until initial set begins, quickly remove and trim undercuts—rinse with cold water—reinsert for final setting.

DISCUSSION

There is little doubt that the number of bruxism cases has increased in recent years; as has our knowledge and understanding of its diagnosis and the seriousness of the damage it may cause to the masticatory apparatus. Many of the Temporo-mandibular Joint Syndrome cases have been found to be directly related to bruxism. In periodontal disease involving loss of alveolar support, it is generally conceded that the damaging influence of secondary occlusal trauma from bruxism is considerable. The so-called "occlusal equilibration," so widely used today, is not a solution to many of these problems. Neither is an expensive full mouth reconstruction effort always successful.

The possibility that a simple, inexpensive, protective splint may be effective is based on the present view that the preprioceptors in the periodontal ligament play an important role in the neuro-muscular syndrome of Bruxism.

Fig. 8. Splint polished and inserted.

Effect of Somatotropic Hormone on Gingival Wounds in Normal and Protein Deprived Rats

by S. S. STAHL, *New York, New York*

LOCAL administration of somatotropic hormone (STH) has increased the healing rate of experimentally induced wounds by stimulating fibroblasts and granulation tissue formation.[1,2,3,4,5] A stimulating effect on non-injured oral tissues has also been described in normal and nutritionally depleted animals.[6,7,8,9] The present study deals with the response of injured periodontal tissues to the local application of STH.

MATERIAL AND METHODS

Fifty-five Long-Evans strain, male rats weighing an average of 230 gms. at the beginning of the experiment were divided into the following groups:

Group 1. 10 animals received injections of 0.5 mg. of STH in 0.1 cc. of distilled water buffered to a pH of about 10 three times weekly over a period of six weeks.

Group 2. 5 animals received injections of 0.1 cc. of distilled water buffered to a pH of about 10 in a manner similar to that described for Group 1.

Group 3. 10 animals received an injury to the interproximal gingiva between the first and second left maxillary molars at the beginning of the experiment and then received STH injections in a manner similar to that described for Group 1.

Group 4. 5 animals received a gingival injury similar to that induced in Group 3 and injections of the vehicle as outlined for Group 2.

Group 5. 15 animals were placed on a protein-free diet* at the beginning of the experiment and then received a gingival injury and STH injections as in Group 3.

Group 6. 10 animals were placed on a protein-free diet at the beginning of the experiment and then received a gingival injury and vehicle injections as in Group 4.

The gingival injury was created by drilling a channel through the interproximal embrasure of the maxillary left first and second molars which removed the gingival papilla and the dental contact point. This led to food impaction and chronic inflammation in all specimens examined.

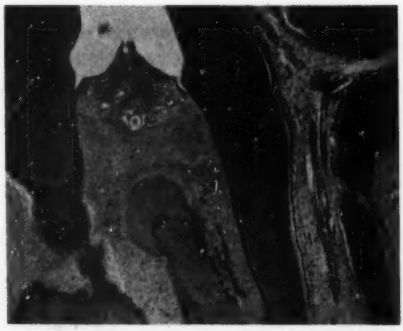

Fig. 1. Photomicrograph of an interproximal papilla, experimental site, of a rat receiving injections of STH into the papilla. H.xE. stain, Magn. 72X. Note the apposition of bone at the alveolar crest.

The site of injection was the interproximal papilla area between the first and second maxillary molars approached palatially in all animals.

All animals received purina chow or the special diet and water ad libitum. At the end of the experimental period, six weeks, the maxillae were dissected out and prepared for histologic examination.

GROSS OBSERVATIONS

Animals receiving STH injections into

From the Murry and Leonie Guggenheim Foundation, Institute for Dental Research and the Department of Periodontia and Oral Medicine, New York University College of Dentistry, New York, New York.

This study was supported in part by grant-in-aid D-537 from the National Institute for Dental Research of the National Institutes of Health, U.S. Public Health Service, Bethesda, Maryland. The STH was supplied by a grant from the Endocrinology Study Section of the National Institutes of Health, Bethesda, Maryland. Presented in abstract at the 37th meeting of the International Association for Dental Research, March 20, 1959, San Francisco, California.

*The protein-free diet was obtained from Nutritional Biochemical Company, Cleveland, Ohio.

the papilla, Group 1, gained weight more rapidly (average weight gain = 79 gms. ± 10.3) during the experimental period than did animals receiving injections of the vehicle, Group 2, (average weight gain = 50 gms. ± 13.3).

Animals receiving a gingival injury in addition to local injections of STH, Group 3, gained less weight than their respective controls, Group 1. (Average weight gain Group 3 = 59 gms. ± 10.4).

Animals on a protein-free diet and receiving gingival injuries lost approximately half of their initial weight during the experimental period. STH injections in these animals did not alter the weight loss.

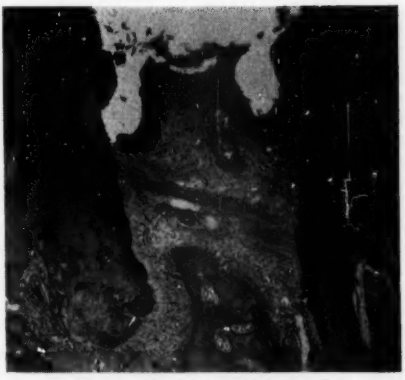

Fig. 3. Photomicrograph of an injured interproximal papilla of a rat receiving STH injections into the papilla. H.xE. stain, Magn. 50X. Note the marked osteogenic activity at the crest.

Fig. 2. Photomicrograph of an interproximal papilla, experimental site, of a rat receiving injections of the vehicle into the papilla. H.xE. stain, Magn. 72X. Note the apposition of bone at the crest.

HISTOLOGIC OBSERVATIONS

The experimental site in all animals was the interproximal papilla between the first and second maxillary molars. For comparative purposes, this area will be described throughout the study.

Animals receiving injections into the interdental papilla showed food impaction associated with a subepithelial inflammatory infiltrate. Apposition was noted at the alveolar crest and was uniformly present in both STH and vehicle injected animals (Figure 1 and 2). No difference was noted in the periodontal membrane between the hormone injected and control groups.

All animals receiving a dental injury showed a marked gingival recession. The epithelial layer appeared thickened and was often keratinized. A subepithelial inflammatory infiltrate and increased vascularity were frequently noted. Transeptal fibers were present below the inflammatory infiltrate.

Animals receiving STH injections into the site of injury showed marked interdental crestal apposition in most specimens studied. Increased osteoblastic activity was

Fig. 4. Photomicrograph of an injured interproximal papilla of a rat receiving STH injections into the papilla, H.xE. stain, Magn. 50X. Note the new bone forming at the crest.

Fig. 5. Photomicrograph of an injured interproximal papilla of a rat receiving injections of the vehicle into the papilla. H.xE. stain, Magn. 50X. No osteogenic activity is noted at the crest.

seen surrounding the interdental alveolar septum (Figure 3 and 4). Vehicle injected animals showed little evidence of apposition at the crest and appeared similar to injured, non-injected specimens (Figure 5 and 6).

Silver impregnation showed a greater amount of argyrophilic fibers in the transseptal fiber groups in STH injected animals than in the control groups.

Animals on a protein-free diet receiving a gingival injury and injections of the vehicle showed a gingival recession with resorption at the interdental crest (Figure 7). The periodontal membrane exhibited a reduction in cellularity. A marked osteoporosis was also noted in the interradicular septi (Figure 8).

Animals on a protein-free diet receiving a gingival injury and STH injections also showed a marked gingival recession. The periodontal membrane in these specimens appeared to be more cellular than was seen in the control group described above. The interradicular septi showed endosteal apposition (Figure 9). Apposition was also noted at the periodontal surfaces and at the interdental alveolar crest (Figure 10). The osteogenic activity at the crest in these animals however, was much less prominent than observed in normally fed animals receiving a gingival injury and STH injections.

Fig. 7. Photomicrograph of an injured interproximal papilla of a rat on a protein deprived diet, receiving injections of the vehicle into the papilla. H.xE. stain, Magn. 60X. Note that despite the high coronally placed dental injury, resorption at the crest is present.

Fig. 6. Photomicrograph of an injured interproximal papilla of a rat not receiving any injections in the area. H.xE. stain, Magn. 50X. Little osteogenic activity is seen at the crest.

COMMENT

Repeated observations have demonstrated the ability of STH to increase wound healing and granulation tissue formation in experimentally created skin wounds.[1,2,3] A rationale for this action has been suggested through a direct stimulating effect on fibroblastic activity at the site of injury[1,3] and a general nitrogen retention without

significant weight gains which occurs following STH administration.[10,11]

Dental injuries created under the described experimental conditions and due to the specific environments at the site of injury, presented a problem in healing somewhat different from that encountered in skin injuries. The dental wound affected both soft and hard tissues in addition to specific tissue relationships such as the epithelial and periodontal attachment apparatus. An additional factor present was the continuous food impaction at the site of injury which acted as a chronic irritant. It was of interest to note that STH, under these circumstances, was unable to prevent gingival recession with the concomitant resorption of the alveolar crest and could not induce periodontal fiber reattachment at the level present prior to injury. STH injections, however, were able to create an environment which appeared more favorable to crestal osteogenesis at the site of injury than was present in the control animals. The specific mode of STH action under these circumstances has not been determined.

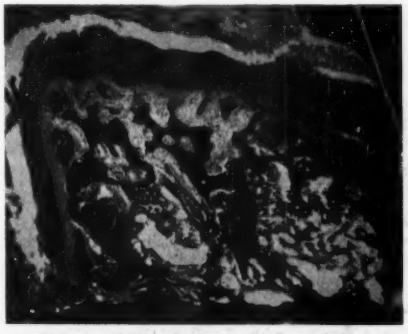

Fig. 8. Photomicrograph of an interradicular septum of a rat on a protein deprived diet, receiving injections of the vehicle into the papilla. H.xE. stain, Magn. 60X. Note the marked endosteal osteoporosis.

Of further interest was the observation regarding the increased cellularity of the periodontal membrane and the inhibition of the endosteal osteoporosis in STH treated animals on a protein-free diet as compared with the endosteal osteoporosis seen in the protein-free fed vehicle injected group. These findings are similar to those reported for non-injured, STH treated animals on nutritionally deficient diets[8,9] and may be the result of the nitrogen sparing action of STH even in nutritionally deficient animals.[10]

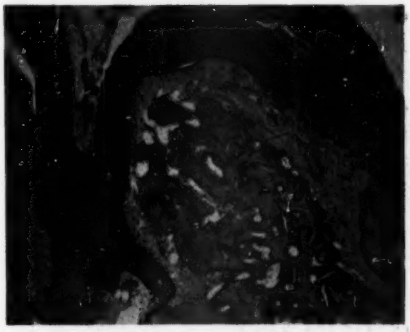

Fig. 9. Photomicrograph of an interradicular septum of a rat on a protein deprived diet receiving injections of STH into the papilla, H.xE. stain, Magn. 75X. Note the normal appearance of the septum with evidence of endosteal apposition.

SUMMARY AND CONCLUSIONS

Gingival injuries were experimentally created in the interdental papilla between the left first and second maxillary molars

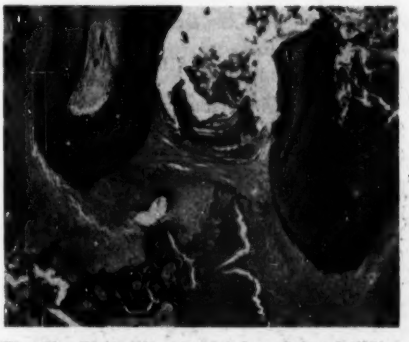

Fig. 10. Photomicrograph of an injured, interproximal papilla of a rat on a protein deprived diet, receiving injections of STH into the papilla. H.xE. stain, Magn. 60X. Note that despite the apical level of the injury with gingival recession, some apposition can be observed in areas of the alveolar crest.

of young, adult male rats. STH was injected directly into the site of injury.

Increased osteogenic activity at the alveolar crest of the injured area was noted more frequently in the STH treated animals than in the animals receiving injections of the vehicle.

Protein deprivation was superimposed upon a group of animals receiving the gingival injury as well as STH or vehicle injections. STH treated animals did not exhibit the reduction in cellularity of the periodontal membrane and endosteal osteoporosis seen in the control animals and in some specimens showed osteogenic activity at the periodontal surfaces of the interdental and interproximal septi.

The author wishes to acknowledge the technical assistance of Marcella Hazan, Dr. Biol. and Helen Rosenman, B.A., in the preparation of this study.

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Book Notice . . . .

AN INTRODUCTION TO PERIODONTIA

By Henry M. Goldman, D.M.D., F.A.C.D., Professor and Chairman, Department of Stomatology, Boston University School of Medicine; Saul Schluger, D.D.S., Director of Graduate Education and Professor of Periodontology, University of Washington School of Dentistry; D. Walter Cohen, D.D.S., Assistant Professor of Oral Medicine and Histopathology, University of Pennsylvania School of Dentistry; Bernard Chaiken, D.M.D., F.A.C.D., Head of Periodontia Department, Riesman Dental Clinic, Beth Israel Hospital, Boston, and Lewis Fox, D.D.S., F.A.C.D., Associate Professor of Periodontology, University of Pennsylvania Graduate School of Medicine. 180 illustrations—348 pages. $7.75. February, 1959.

This concise pocket-sized book is an excellent summary of periodontology. Its illustrations are well chosen and of high quality. The book considers periodontal disease manifestation, examination, diagnosis, and treatment, and would serve as an excellent introduction or review of periodontology.

A Permanent Fixed Amalgam Splint

by RALPH S. LLOYD, D.D.S.* AND PAUL N. BAER, D.D.S.,** *Bethesda, Md.*

IT IS frequently necessary to construct a permanent, fixed splint, as part of periodontal therapy, in a mouth with gross loss of attachment apparatus. Since the rationale for the use of such splints has been adequately covered in several recent textbooks (1, 2), a discussion of this phase will be omitted from this paper. This report will present and describe the construction of a matrix with which physiologic interproximal contours can be obtained in a continuous amalgam splint.

The advantages of this type of splint over those of the more conventional gold, or combination gold and acrylic, are as follows:

1. It is more economical in time and materials.

2. The teeth do not have to be in perfect alignment, as parallelism is not necessary.

3. Perfect margins are easier to obtain.

4. It can frequently be made more esthetic than most other types of splints.

Its chief disadvantages are:

1. In patients with a high caries rate, it does not afford the protection of full coverage.

2. Its use is limited to posterior teeth.

LABORATORY PROCEDURES

A. Preparation of Models.

Amalgam cavity preparations are made in the teeth to be splinted, Figure 1. A rubber base impression is taken of these and a cast is poured in stone. Wax is inserted into the cavity preparations of the cast and physiologic interproximal contours carved, Figure 2. The entire stone cast is then coated with a separating medium, such as liquid foil. Finally, plasticine is placed, from the lingual, to fill in one-half of the interproximal spaces. The buccal portion of the interproximal area, however, must be kept free and clear.

Fig. 1. An example of the type of amalgam preparations used.

B. Construction of Hinge.

A .040" stainless steel wire, 3 inches in length, is bent double onto itself. On the outside of the bend, a 5 mm. length of tube, .040 internal diameter, is soldered. Another .040 wire of similar length is then threaded through the inside of this tube and bent double onto itself. This forms a hinge, Figure 3. The free ends of both .040 wires may be soldered together. The hinge is then placed distally around the last tooth in the segment to be splinted, and adapted to the buccal and lingual surfaces of the teeth.

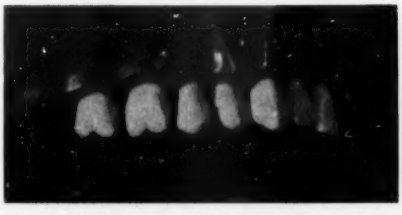

Fig. 2. Wax is inserted into the cavity preparation and physiologic interproximal contours carved.

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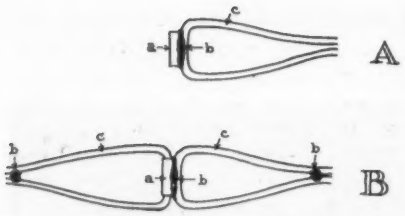

Fig. 3. Method for construction of hinge:
a. tube of .040 internal diameter
b. solder
c. .040 wire

Fig. 4. The buccal and lingual acrylic segments are attached distally by the hinge. Small acrylic spines are projecting interproximally.

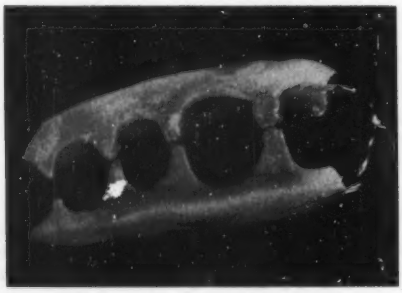

Fig. 5. A gingival view of the spine-like projections which will form a matrix for the interproximal contours.

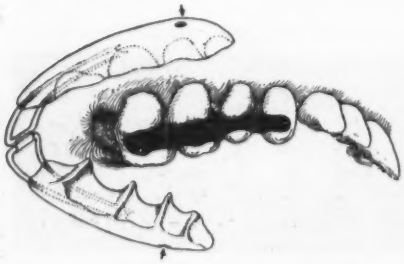

Fig. 6. Arrows indicate indentations which are made in acrylic matrix to receive the teeth of the No. 1 amalgam matrix retainer.

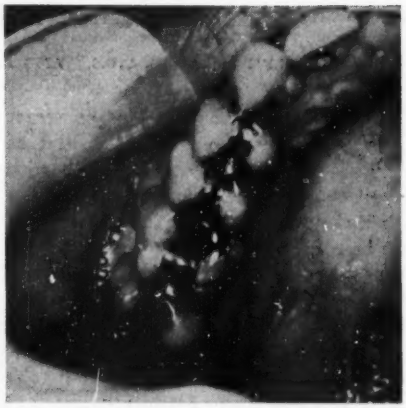

Fig. 7. An occlusal view of an amalgam splint which extends from the maxillary 1st bicuspid to the maxillary 2nd molar.

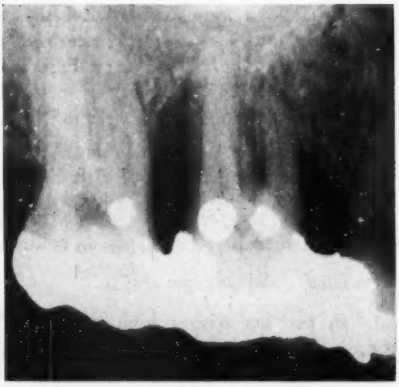

Fig. 8. This is a radiograph of the amalgam splint shown in Fig. 7.

Fig. 9. A buccal view of the splint shown in Fig. 7.

C. Processing of acrylic matrix.

Self-curing acrylic is painted over the wires and around the teeth on the buccal aspect only, taking care that the acrylic goes through the interproximal spaces. The hinge must be left uncovered. The plasticine is then removed from the lingual interproximals. Separating medium is reapplied and the above procedure repeated on the lingual aspect. Upon removing the splint from the stone cast, it will be seen that the buccal and lingual acrylic segments are attached distally by the hinge, and small acrylic spines will be seen projecting interproximally, Figures 4 and 5. These spine-like projections will form a matrix for the interproximal contours of the amalgam splint.

CLINICAL PROCEDURES

The acrylic matrix is inserted into the patient's mouth and held in place with a No. 1 amalgam matrix retainer, Figure 6. By condensing amalgam filling material into all the cavity preparations, a one piece amalgam periodontal splint is formed. When the initial set has taken place, the matrix is removed, and the individual occlusal contours carved, Figure 6, 7. The acrylic matrix will have helped to contour the interproximals and to have helped prevent the formation of overhanging filling material. Final finishing of the interproximal contours done by means of hand instruments and the use of fine, wet, linen sandpaper strips, will produce the results shown in Figure 8. A view of the buccal aspect of a splint which is continuous from the maxillary right 1st bicuspid to the maxillary right 2nd molar Figure 9, demonstrates the minimal exposure of filling material and resulting good esthetic effect.

In cooperation with the Bureau of Standards, we are now testing various implant wires for reinforcement. These implants are made of silver, stainless steel, and gold.

SUMMARY

It has been demonstrated that immobilization of teeth is a very beneficial procedure in periodontal therapy. The amalgam splint described provides an easy, inexpensive, and efficacious method of immobilizing posterior teeth.

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ARTHUR F. LUFKIN, D.D.S.

1889-1959

It has just come to our attention that Dr. Arthur W. Lufkin of Hollywood, California, died on January 15, 1959. Dr. Lufkin is well-known in the fields of dentistry because of his many publications and his activities in the field of oral pathology and oral histology. He was a Diplomate of the American Board of Periodontology and was elected to honorary membership in the American Academy of Periodontology on November 8, 1958. He served as President of the Los Angeles County Dental Association and then was editor of the Journal of the Southern California State Dental Association for 12 years. He was given the Los Angeles County Dental Association plaque for achievement in 1949.

Announcements . . . .

THE AMERICAN ACADEMY OF PERIODONTOLOGY

The American Academy of Periodontology will hold its Annual Meeting at the Barbizon Plaza Hotel, 106 Central Park South, New York City, on September 10th, 11th and 12th, 1959.

This year's meeting will score a first in the presentation of clinical material, inasmuch as clinics will be presented from the Walter Reed Army Medical Center, and the Armed Forces Institute of Pathology in Washington, D. C.

These clinics will be presented by means of closed circuit colored telecasts with wide screen projection in the Barbizon Plaza Hotel, New York City.

Participation in this meeting is open to all members of the American Dental Association upon the payment of a modest registration fee.

BAYLOR UNIVERSITY COLLEGE OF DENTISTRY

The division of graduate studies, Baylor University College of Dentistry, Dallas, Texas, announces the following advanced courses of study in Periodontology, beginning in September, 1959:

1. A nine-month course of postgraduate study leading to the degree of Bachelor of Science in Dentistry. This course consists approximately half of advanced study in basic science and half of clinical study.

2. A twenty-month course (two regular school terms and one summer session) of graduate study leading to the degree of Master of Science in Dentistry. This consists essentially of the B.S.D. course above, followed by a summer term of clinical experience and then a second full nine-month term of advanced study in theory and clinical practice. A thesis is required.

Baylor has a modern school plant fully equipped for undergraduate and graduate study and completely air-conditioned. Every opportunity is offered the graduate student to prepare himself for the practice of Periodontology. For further information, write to the Registrar, Baylor University College of Dentistry, 800 Hall Street, Dallas 10, Texas.

10th ANNIVERSARY BERKSHIRE CONFERENCE

The 10th annual Berkshire Conference will be held at Easton in Lenox, Massachusetts, June 14 to 18, 1959. The subject will be "Trauma and Oral Disease" and the speakers will include Dr. Balint J. Orban, Dr. Milton B. Engel, Dr. Basil G. Bibby, Dr. Joseph L. Bernier, Dr. Louis Weinstein, Dr. Sigurd P. Ramfjord and Dr. Irving Glickman. This popular Conference is always well attended so those desiring to go should make their reservations at once. Information may be obtained by writing Dr. Glickman, Director of the Division of Graduate and Postgraduate Studies, 136 Harrison Street, Boston, Massachusetts.

Plan NOW to attend the Centennial Session of the American Dental Association, New York, N. Y. Sept. 14-18, 1959. The 100th anniversary meeting, combined with the 47th annual session of the Fédération Dentaire Internationale, promises to be the most stimulating dental convention of the century!

Bring the family and combine a well-deserved vacation in the world's most exciting city with your participation in an outstanding scientific meeting. Use the handy application blank in the current issue of THE JOURNAL OF THE AMERICAN DENTAL ASSOCIATION to reserve the hotel accommodations of your choice.

TUFTS UNIVERSITY

A postgraduate course in *Cosmetic Variations in Jacket Crown and Bridge Restorations Involving Periodontal Lesions* will be given Monday and Tuesday, September 21 and 22, 1959 by Dr. Charles L. Pincus, D.D.S., Beverly Hills, California. This course will cover the technical procedures presented in multiple jacket crown and bridge restorations with special emphasis on cosmetics. Tooth preparations will be discussed which will assist in securing desirable functional and cosmetic results with special consideration given to maintenance of good gingival and periodontal health. Tuition $60.00—Class limited.

For further information and application write to Director, Division of Graduate and Postgraduate Studies, Tufts University School of Dental Medicine, 136 Harrison Avenue, Boston, Massachusetts.

OPEN LETTER FROM THE PRESIDENT OF THE AMERICAN ACADEMY OF PERIODONTOLOGY:

Because of the special significance of our annual meeting to be held in New York City September 9-12, 1959, I thought it worthwhile to report something of our plans.

This being the centennial anniversary of the American Dental Association, we are planning some unique activities in which I am certain you will be interested. The program will have an international flavor and many of our fellow workers from foreign countries will be here at that time. You will be particularly interested in the panel discussion which will deal with some of the research and forms of treatment that have gained popularity in other lands.

A unique feature of the program will be a live television presentation which will emanate from the Armed Forces Institute of Pathology in Washington, D. C. and be displayed as part of our scientific session. This program will be in color and will be viewed on a theatre size screen. This will be an interesting and rewarding experience.

The balance of the program has been carefully arranged by your able program committee under the chairmanship of Doctor Irving Glickman. In it there will be a most illuminating group of presentations on modern periodontal problems.

Our hotel, the Barbizon Plaza, is ideally situated near Central Park. It is close to the auditorium where the American Dental Association activities will be centered and is convenient to all parts of the city. We anticipate a large attendance because of the centennial aspects of the occasion and I would therefore urge you to make early reservations.

Our annual banquet, as well as the round table luncheon will also be something out of the ordinary. An outstanding program has been arranged for both which will be of great interest to you. All in all, we are anticipating an exceptional meeting which you will, no doubt, thoroughly enjoy.

Joseph L. Bernier, President

P. S. Dr. Norman Vincent Peale has accepted the invitation to speak at the banquet honoring his esteemed friend, Dr. Arthur H. Merritt.